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TUBERCULOSIS OF THE LUNGS AND BRONCHIAL GLANDS IN CHILDREN.[1]

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IN THE MEDICAL JOURNAL OF AUSTRALIA of June 21, 1930, there is an article by Dr. Wilfred Evans,(1) of Sydney, on pulmonary tuberculosis in childhood, with special reference to its radiological aspect. After discussion of the subject he draws several conclusions, the first of which is as follows: "Tuberculous disease in young children is suspected more frequently than it exists."

Apropos of this, it may be argued that tuberculous infection does not necessarily mean that a child so infected is suffering from tuberculosis. The great difficulty is to know where to draw the line. I am of the opinion that tuberculous infection, however small the lesion be, which is giving rise to any symptoms of ill health, should be regarded as active and dangerous tuberculous disease and treated accordingly. It is for this reason that I contest the statement which I have just quoted, and affirm that in South Australia, tuberculosis in children is much more common than is generally believed, and I consider that the same will apply to any country or State in which pulmonary tuberculosis in the adult is as widespread as it is in this State.

The prevention, diagnosis and treatment of tuberculosis is one of the greatest problems of medicine.

Infants and young children are notoriously susceptible to infection with the tubercle bacillus. Could the dose of infection and the virulence of the infecting organism be satisfactorily regulated, it is possible that the ideal condition of immunity could be arrived at. The chief danger lies in massive infection, and this is the risk which we as a profession should seek to minimize.

[1] Read at a meeting of the South Australian Branch of the British Medical Association on September 25, 1930.

Tuberculosis is not an hereditary disease, though one does come across the very rare case of the tuberculous infant which has been infected from the maternal blood stream through the placenta, the normal barrier being overcome by the existence of some local lesion. This happening is so rare that for all practical purposes it can be ignored and attention concentrated on acquired infection.

In Great Britain infection by the bovine type of tubercle bacillus ingested in milk from a tuberculous cow appears to be much more common than it is in this country, where infection with the human type of bacillus is the rule.

It is for this reason that I would particularly stress the history of contact of the child suspected of infection, with a tuberculous relative. This contact needs to be intimate and prolonged. The most common examples are shown by children infected by a tuberculous mother or father, though one sees instances where some tuberculous relative or friend has been living under the same roof as the sick child.

A common story is as follows. The child is brought by a parent, complaining that the child is easily tired, wishes to lie down rather than play with others, that he has a poor appetite and picks at his food, that he is not gaining weight or, perhaps, that he has lost weight. At times he is noticed to be feverish, and he may have a short dry cough. This is an average story, but the symptoms may be less.

The parents are then questioned as to the previous health of the patient. Note is taken of the occurrence of whooping cough. A careful family history is then taken and, here is the most important point of the whole examination, an endeavour is made to ascertain whether the child has been in close contact with any person who may be suffering from tuberculous disease. Far too often, unfortunately, the answer is that the father or the mother has pulmonary tuberculosis, or is suspected of having such. At times a relative similarly affected has lived under the same roof. In one extreme case which I have been told of, the patient had slept in the same bed as an uncle dying of tuberculosis. The answer is sometimes more vague. It may be that a grandmother living with them has had "bronchitis" for years or that the father suffers from "asthma," but has not consulted a doctor. Again, fortunately, the parent can very frequently affirm that there has been no contact with tuberculosis; I use the word "contact" here in the sense of prolonged intimate contact. In such instances the chances are very much against the child's present condition being due to tuberculosis.

The next step comprises the ordinary physical examination. Particular note is taken of the presence or absence of tonsillar sepsis, of glandular enlargement, mouth breathing, nasal discharge and clubbing of the fingers. A careful examination of the chest is conducted in a good light. The presence of enlarged veins in the thoracic wall is of some importance. Signs of gross enlargement of the bronchial glands as paravertebral dulness, d'Espigne's sign and high-pitched breath sounds in the axillary region are not commonly discovered. Frequently the physical examination gives one no clue, but the absence of physical signs does not mean the absence of disease. That is a remark on which I wish to lay particular emphasis.

The performance of a tuberculin test comes next, and the one I have been in the habit of using is the Mantoux intradermal test. One-tenth of a cubic centimetre of a one in a thousand freshly prepared dilution of old tuberculin (Commonwealth Serum Laboratories) is injected into the skin of the anterior surface of the forearm. The Mantoux test is used in preference to the von Pirquet test because it is more easily and rapidly performed, and there is no need for a control test. With the use of a sharp, short-bevelled needle it is practically painless. The average child is interested in watching it done. There are no dangers and no drawbacks attached to this test. The only sequelæ I have seen have been the persistence of a small papule at the site of the injection on two occasions when there had been a positive reaction. These papules are apparently in the nature of a tuberculide. The parent is told to bring the child in three days' time, when the result of the test is read and recorded in a book. This test is found to be of great value in investigation of sickness in children. For some time physicians at the Out-Patient Department of the Children's Hospital, inspired by Dr. E. Britten Jones, who took the lead in the matter, have conducted the Mantoux test on numerous new patients, irrespective of their complaint. The results are interesting, as I shall show you.

The total number of children tested was 406; all were under the age of twelve years. Of these there were 75 with a history of contact, of whom 72% gave a positive reaction. There were 23 with doubtful history of contact, of whom 61% gave a positive reaction. There were 308 with no history of contact, of whom 5% gave a positive reaction. Or, looking at it in another way, of 84 positive reactors there was a history of contact in 63% and of possible contact in a further 16%, leaving 21% with no history of contact.

Of those who failed to react, 322 in number, there was no history of contact in 90%, history of contact in 6%, and history of possible contact in 4%.

Since writing these notes my attention has been drawn to an article by Schlesinger and Hart.[2] Conclusions drawn from an investigation conducted by these men at an English hospital are very similar to those I am placing before you.

Should there be no history of contact with tuberculosis and the Mantoux reaction be absent, one can be fairly certain that the child's present condition is not due to tuberculous infection. Should there be a history of contact and the skin fail to react to the Mantoux test, the test is repeated with a dilution of one in 100 of old tuberculin. It is unusual, however, for the reaction to be absent when there is a definite history of contact.

The next step in the investigation is carried out in the Radiological Department, where the chest is screened and pictures taken in both antero-posterior and lateral positions. In positive reactors it is usual to find confirmatory X ray evidence of tuberculous disease in one or both lungs or in the bronchial glands, more commonly the latter. Often evidence of calcareous change is detected in the bronchial or paratracheal glands. The latter is best demonstrated in the lateral view.

It has happened that the tuberculous focus cannot be shown in the chest photographs; it may then possibly be demonstrated by a picture of the abdomen when calcareous changes in the abdominal glands may be revealed.

Should these investigations lead one to suppose that the child is suffering from a tuberculous infection he is admitted to hospital for further observation. Careful temperature records (and for this purpose an axillary temperature is worthless) are charted every four hours.

It is unusual to see the adult type of pulmonary tuberculosis in children, although it does occur, as you have seen in the patient shown by Dr. Britten Jones tonight.

It is also unusual to obtain sputum from these children. The sick child with copious sputum is generally suffering from another complaint, bronchiectasis secondary to sinus and tonsillar disease, of which more anon.

Mode of Production of Lesions.

The commonest method by which lesions are produced is inhalation of tubercle bacilli with the formation of the so-called primary focus in the lung, a nodule of infiltration, most commonly in the lower part of the lung. This primary focus may be so minute as to defy detection and may be difficult to demonstrate at autopsy. From this primary focus the infection spreads by the lymphatics to the bronchial glands, draining the affected part. Once established in this way, the process may become quiescent and heal or, if resistance be poor, advance. In cases of progressive disease the great danger is from involvement of a neighbouring vein in the tuberculous process, for an intimal tubercle may form and when this latter breaks down, the infection becomes disseminated by the blood stream, giving rise to acute miliary tuberculosis which is invariably fatal. The common ending in cases of this nature in children is death from tuberculous meningitis.

In the chest of a tuberculous child one does not expect to find physical signs which are in any way comparable with those found in the adult suffering from pulmonary tuberculosis. The latter type of patient has had a primary infection in childhood, and this infection has followed the usual course of secondary glandular involvement which has become quiescent; this process has produced a relative immunity of the body.

Later in life, during adolescence most commonly, the resistance of the body has been lowered by some means and either by the advent of fresh infection or by the reactivation of the hitherto dormant primary lesion, a second active tuberculous process has arisen. The response in the adolescent is different from that in the child, for in the former we find infiltration, cavitation, fibrosis *et cetera*, as opposed to the common glandular involvement with possibility of infection of bones and joints in the latter.

It has been stated by Bacmeister that for the production of typical phthisis a relative immunity of the body is necessary, and this is produced by an early primary infection.

Differential Diagnosis.

The condition which is most commonly confused with tuberculosis in children is that of chronic bronchitis or bronchiectasis secondary to tonsillar or nasal sepsis or to sinus infection, or to all of these.

I frequently see children with this condition who are brought by an anxious parent whose fear is that they have tuberculosis on account of a cough of months' or years' duration, with the presence of sputum and possibly accompanied by night sweats. The latter symptom is especially dreaded by the laity.

In this condition there is nearly always no response to the Mantoux test. The X ray picture shows changes which are more marked at the bases of the lungs, especially the left lung. The actual bronchiectatic dilatations can be outlined by the use of an intratracheal injection of "Lipiodol" prior to the taking of the skiagram. This manœuvre is not absolutely devoid of risk. General anæsthesia carefully controlled is necessary. Recently the use of rectal injection of "Avertin" has been tried for the production of anæsthesia in these cases with, I understand, satisfactory results.

As a good example of bronchiectasis following tonsillar and antral sepsis I may quote the condition of the patient which I now describe.

A.B. was first seen in 1924 when he was seven years old. The notes state that the boy's tonsils were enlarged and reddened, and there was a chronic nasal discharge. X ray examination revealed opacity of both maxillary antra and of the left frontal sinus. He was referred to the Ear, Nose and Throat Department for operation, but the advice was not heeded and he disappeared for four years. When next seen, he gave a history of cough of three years' duration. This cough was accompanied by copious purulent sputum which was sometimes streaked with blood. He was undersized, languid and somewhat deaf. He had a poor appetite and was not gaining weight. There was no history of contact with tuberculosis. There was chronic nasal discharge, tonsillar sepsis, clubbing of the fingers. The percussion note was impaired at the base of the left lung posteriorly, with râles to be heard in that situation. There was no reaction to the Mantoux test. No tubercle bacilli could be demonstrated in the sputum. He was treated by having the maxillary antra drained and the tonsils and adenoids removed. He has gained weight and looks much better, but the damage to the lung is considerable and he will always have a cough with sputum.

Confirmatory "Lipiodol" injection in this case was not permitted by the parents.

The parents of this boy had previously been told that he was suffering from tuberculosis, for which they were advised to secure his admission to a sanatorium, which was the very thing one should avoid, for he would there be brought into contact with people with open tuberculosis. I am not suggesting that he would have been accepted for

admission by the medical officer in charge of any sanatorium.

Another condition which is apt to mimic the symptoms of latent or smouldering tuberculosis in children is that of chronic intestinal indigestion. Children so afflicted are languid, pick at their food, have occasional bouts of feverishness, look ill, and gain weight slowly. Here again the Mantoux test is a very good guide, though it must be realized that the child may be suffering from both tuberculosis and chronic intestinal indigestion.

Another common cause of loss of appetite, failure to gain weight, and bouts of pyrexia, is chronic pyelitis.

Treatment.

Prophylaxis.

Obviously the most important part of treatment is prophylaxis. Prevention of infection with the bovine tubercle bacillus is a simple matter. Tuberculin testing of cows plays its part, but safety can be obtained by the boiling of all milk used, for three minutes. In this country there is far greater danger from diarrhœal diseases caused by contaminated milk than from any other form of milk infection, so that the boiling of milk serves a useful purpose, even when there is no infection with tubercle bacilli.

Prevention of gross or massive infection with the human tubercle bacillus is much more difficult, and here it is that the family doctor can be of such assistance. You must all of you have in your practices one or more adults suffering from open pulmonary tuberculosis. Any children living in the same house or visiting the house frequently, are in grave danger of becoming infected. I consider that children who are contacts should be under periodical medical supervision. If possible, a complete examination of each such child should be carried out, with tuberculin testing, as outlined above. In France there is a scheme known as the Grancher system, whereby the children from infected households are boarded out to healthy foster parents, a plan which it would be difficult to bring into operation in this country. The best that we can do at present, is to insure that all precautions should be taken with the sick person. These precautions are too well known to need recapitulation. If the children can be kept from contact with the infected person or his belongings, and kept from entering his bedroom, and if he exercise every precaution, especially as regards disposal of sputum, the chances of their acquiring infection are very much lessened. It has been shown in the Papworth colony in England that healthy children can be raised by tuberculous parents who have been correctly trained.

Treatment of Infected Children.

In regard to treatment of the child suffering from tuberculous infection, when there is pyrexia, the child should be kept at absolute rest in bed in the open air. This is not so easy as it sounds, as these young patients soon start to feel well again and promptly indulge in numerous activities, so that at times some form of restraining apparatus may become necessary. A good, nourishing, plain diet with fresh vegetables, fruit, extra milk, butter and cream is ordered.

Cod liver oil in some form is of assistance, always provided that it does not interfere with the digestion. It is better omitted during the hot summer months. As the temperature settles down, more activity is allowed just as in the case of the infected adult. Finally the child reaches the stage when ambulatory treatment can be instituted. Change of air plays its part. I find that during about eight months of the year these children do well when sent to Mount Lofty.

In cases where one lung is extensively involved and the other lung is clear or but slightly affected, the induction of an artificial pneumothorax offers the best chance of arrest of the disease. Children stand this well, especially if given a small dose of morphine prior to the initial introduction of the trochar. There are times when it is found impossible to secure any or more than partial collapse of the lung. I have seen one patient who had pleural adhesions which were too extensive to allow of collapse. We can then fall back on the operations of phrenic avulsion and thoracoplasty, a very good example of which has been demonstrated to you tonight and one attended by a good therapeutic result.

This operation should not be performed without very careful consideration, for the diaphragmatic paralysis produced by phrenic avulsion cannot be rectified subsequently.

Use of Tuberculin. I realize that in discussing tuberculin therapy I am entering controversial grounds. My own feeling is that tuberculin, carefully administered, is of greatest assistance to certain types of patient who appear to have poor resistance to the disease and to make no headway towards overcoming it.

I have been in the habit of giving tuberculin by the percutaneous method, namely, by inunction of tuberculin ointment. I have used the product put up by the Commonwealth Serum Laboratories and have found it satisfactory. A very small portion of the ointment, a piece about the size of one-quarter of a pea, is rubbed with a glass rod into the skin of the abdominal wall. Watch must be kept for local, general and focal reaction. Local reaction is the rule, and takes the form of a papular rash at the site of inunction. The reason I start with such a small portion of ointment is on account of the general reaction with pyrexia and malaise which I observed in some children anointed with a larger amount. The inunctions are repeated at weekly intervals.

Admittedly, this is an unscientific method of administration of tuberculin, but I have been favourably impressed with the results in the few cases in which I have used it. These children were making little headway prior to the commencement of the inunctions. Following a course of the inunctions there was a gain in weight and a feeling of well-being as evidenced by the child's behaviour.

I have used subcutaneous injections of tuberculin in certain instances, but always in very minute doses.

Conclusion.

The subject on which I have just touched lightly, is a vast and important one, and I trust that my few remarks may be provocative of some discussion amongst you.

What is the significance of this tuberculin reaction? Is a child, apparently in perfect health, who reacts mildly to tuberculin, in a safer position for the future than a similarly healthy child who does not react?

In the instructive article I have quoted, by Schlesinger and Hart, there appears a chart comparing the tuberculin reactions of non-contacts and of home contacts of pulmonary tuberculosis with those of the general population (hospital class) of London for 1929, all the latter being clinically non-tuberculous. The interesting thing shown here is that contacts yield a positive reaction to the Mantoux test at a much earlier age, a percentage of 78 giving a positive reaction by the age of nine years, as compared with a percentage of 86 by the age of twenty-four years in the general population. The reading for the non-contacts follows the latter percentage very closely.

It would appear that the early appearance of a positive reaction to the tuberculin test is unfavourable as denoting early and massive infection, and the earlier the infection the less the chance of survival.

Acknowledgements.

I have to thank my colleagues at the Adelaide Children's Hospital for their assistance in allowing me access to their clinical records. Especially would I thank Dr. Stanley Verco who has provided the skiagrams I have shown, and Dr. E. Britten Jones for many helpful suggestions.

References.

(1) Wilfred Evans: "Pulmonary Tuberculosis in Childhood, with Special Reference to its Radiological Aspect," THE MEDICAL JOURNAL OF AUSTRALIA, June 21, 1930, page 803.

(2) B. Schlesinger and P. D'A. Hart: "Human Contagion and Tuberculous Infection in Childhood," *Archives of Disease in Childhood*, June, 1930, page 191.

PURPURA, WITH SPECIAL REFERENCE TO THE JUVENILE TYPE AND ITS ÆTIOLOGY.[1]

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PURPURA is defined by Piney as a variety of morbid states which have in common a tendency to the occurrence of hæmorrhage either in the form of petechial spots, purpuric extravasations or ordinary loss of blood from free surfaces.

[1] Read at a meeting of the Pædiatric Section of the New South Wales Branch of the British Medical Association on October 25, 1930.

Classification of Purpura.

Purpura may be classified according to its ætiology as follows:

1. Purpura of known origin and arising in one of the following conditions:
   - (*a*) Acute infectious diseases and in particular cerebro-spinal meningitis, scarlet fever, typhus fever, small pox, and less commonly typhoid fever and measles. Acute streptococcal septicæmias and subacute bacterial endocarditis are also often accompanied by petechial eruptions.
   - (*b*) Poisoning by drugs such as benzol, benzine and less frequently "Salvarsan" and the iodides.
   - (*c*) Certain forms of anæmia, in particular acute lymphatic leuchæmia and aplastic anæmia.
   - (*d*) Chronic intoxications and wasting diseases such as senility, cachexia from any cause or chronic renal disease.
   - (*e*) Jaundice.
   - (*f*) Scurvy.

   Hæmophilia is not included in this classification.
2. Purpura of unknown or doubtful origin.
   - (*a*) Acute thrombocytopenic purpura or Wehrlof's disease.
   - (*b*) The so-called anaphylactoid group, which includes Schönlein's or rheumatic purpura and Henoch's or *purpura abdominalis.*

The Blood Platelets.

The blood platelets form such an essential part of blood coagulation and the prevention of hæmorrhage that I shall briefly review their morphology and functions before discussing the various aspects of purpura.

They are minute discoidal bodies, in size about half the diameter of a red blood cell, and numbering from 250,000 to 300,000 per cubic millimetre of blood. If they come into contact with a water-wettable surface, such as the skin, they disintegrate and liberate a substance, thrombokinase, which in the presence of calcium salts initiates the process of blood coagulation by converting prothrombin into thrombin. Thrombin, when formed, immediately acts on the soluble fibrinogen of the blood plasma and converts it into insoluble fibrin and thus clotting takes place. Another and more modern theory is that the fluidity of the blood is maintained by an anticoagulant material, heparin, and that this is neutralized by a substance called cephalin when platelets are disintegrated.

Whichever theory is correct, we know that the first step in the process of blood coagulation depends on the breaking up of the blood platelets.

The normal coagulation time at room temperature and estimated by the capillary tube is five to ten minutes.

Blood platelets have other important functions in addition to that concerned with blood coagulation: (i) They assist in repairing damage to capillary endothelium and in preventing the escape of blood by adhering in masses to the injured parts and there forming a white thrombus. (ii) They attach themselves to and agglutinate bacilli which gain entrance to the blood stream. (iii) When disintegrated, they have a vaso-constrictor effect on the capillaries.

The bleeding time is the length of time during which a punctured wound continues to bleed, and is regarded as giving evidence of the condition of the capillary wall and the associated blood platelets. It is estimated by drying off with blotting paper a puncture made by a sharp instrument into the skin and noting when the bleeding ceases. The normal bleeding time is one to three minutes and does not run parallel to the coagulation time.

Symptomatology of Purpura in Children.

I shall not deal in this address with purpura which occurs as a complication of some other well recognized condition, but shall confine my remarks to the group described as idiopathic and usually subdivided into two classes: (i) Acute thrombocytopenic purpura, (ii) anaphylactoid purpura.

Acute Thrombocytopenic Purpura.

Acute thrombocytopenic purpura is associated with the name of Wehrlof; the essential feature is said to be an intermittent tendency to bleed for no accountable reason and remissions may occur which last months or years.

With the kind cooperation of the Senior Physician of the Royal Alexandra Hospital for Children, I have had the opportunity of studying twenty-two cases admitted to the hospital during the last four years. From this study emerges the fact that purpura in children, whatever its ætiology, presents a very well defined clinical entity which is seldom seen in adult life except as the complication of some other disease.

The age incidence is striking. Of the twenty-two cases four occurred during the first year of life; three occurred during the second year of life; four occurred during the third year of life; five occurred during the fourth year of life, and one each during the fifth, sixth, seventh, eleventh, twelfth and thirteenth years of life.

Those cases occurring during the years from seven to thirteen were all recurrent; and so the primary cases all developed during the first six years, and of these 90% during the first four years of life.

The clinical features are characteristic. A child usually robust and well developed, with no familial or personal history of hæmorrhagic tendencies, suddenly develops crops of petechiæ or larger subcutaneous ecchymoses whose colour varies from dark blue or green to brown or black, according to their age. These hæmorrhagic spots may be widely distributed or localized in a particular area and the most striking thing is that their situation is frequently determined by exposure to mild traumata which under normal circumstances would be insufficient to produce any noticeable injury. The legs, for instance, are a very common site for ecchymosis on account of their liability to blows.

In one case which I observed, purpura developed during an attack of pertussis and the congestion during the paroxysms resulted in severe conjunctival hæmorrhage and numerous petechiæ scattered over the head and neck. The other purpuric manifestations were melena and ecchymosis in the lower limbs. In another case the mother stated that if she slapped the child, the outline of her hand was distinctly visible on the child's body on the following day. In the more severe cases, however, subcutaneous hæmorrhages occur without obvious traumata, and bleeding takes place from the mucous membranes of the nose, mouth, lungs, stomach, bowel, uterus, kidney or bladder.

Epistaxis in particular is a very common symptom. In some instances hæmorrhage into the tissues may be profuse enough to cause local sloughing or even gangrene of a limb. A case in point is that of a child who was admitted into the Royal Alexandra Hospital for Children in 1927, under the care of Dr. Edgar Stephen. In this instance gangrene of one leg necessitated amputation and extensive sloughing developed in the lumbo-sacral region and the lobe of one ear.

Abdominal pain and vomiting are complained of in some cases and joint pains in others. Acute nephritis, which is sometimes said to be a complication, was not observed in this series.

Physical examination, apart from the purpuric manifestations, usually reveals no abnormality, the spleen can rarely be palpated, and as a rule there is no enlargement of the lymphatic glands or marked rise in temperature.

The acute fulminating cases form a rather sharply defined group, characterized by extensive subcutaneous hæmorrhage, profuse bleeding from the mucous membranes, high temperature, intense anæmia and death in from twenty-four to forty-eight hours. The degree of anæmia necessarily depends on the amount of hæmorrhage which has taken place. The total leucocyte count was surprisingly constant in this series, varying only from 6,000 to 8,000 per cubic millimetre except in the fulminating cases in which it was as high as 40,000 per cubic millimetre. The differential count was variable. In some cases there was a marked relative increase in the lymphocytes, mononuclear and eosinophile cells, but in others there was a preponderance of neutrophile cells. The blood platelets were usually absent or found in very small numbers during the stage of active bleeding.

The coagulation time was practically always within normal limits—five to ten minutes when estimated by the capillary tube at room temperature. The bleeding time in the active stage was almost invariably increased and sometimes exceeded twenty minutes.

The capillary resistance test of Rumpel Zeide was seldom carried out, as it does not meet with the approval of young patients and little advantage appears to accrue from it.

The course of the disease varies. In the milder cases the first crop of hæmorrhages is the most severe and, if the child is put to bed and protected from trauma, an occasional fresh bruise may appear, but the condition becomes quiescent in a few days. In the more severe cases bleeding continues both subcutaneously and from the mucous membranes for a variable period, but as a rule ceases within a fortnight if no fresh injuries are received. Platelets usually reappear in the blood stream and the bleeding time shortens as the hæmorrhages cease.

In the fulminating cases, as I have stated, death occurs within twenty-four to forty-eight hours. In this series five patients, or 23% of the total, died, four of them within forty-eight hours of admission. I have been able to follow up the later history of nine patients who recovered, with the kind permission of the senior physicians of the Royal Alexandra Hospital for Children and the willing assistance of Dr. Noad and Dr. Bradley in performing the pathological investigations. The result is as follows:

Four of these patients had no definite recurrence of symptoms in the comparatively short period since their discharge, which ranges from nine months to three years.

Three others are subject to recurring attacks of purpura and are unable to lead an active life or indulge in sport, because any mild injury is followed by extensive bruising.

The two remaining patients had been subjected to splenectomy. Since then they have been perfectly well and able to play even football without suffering any ill effects.

The blood counts of eight of these patients, taken in July, 1930, are as shown in Table I.

The results are confusing and are worthy of some comment. The platelet count in all cases is low, but there is no apparent correlation between the number of platelets and the tendency to recurrent hæmorrhages. For instance, the highest platelet count in the series is found in the patient with the greatest tendency to bleeding and the lowest count in patients who had no sign of recurrence.

This suggests that platelet counts in the intervening time between attacks give little guide to the possibility of recurrence. Indeed, throughout all these investigations it has been found that these counts vary a great deal, even in the same individual, and one wonders whether the present methods of estimation are sufficiently accurate.

During an attack of active bleeding, however, the results are very definite; platelets are then entirely absent or present in only very small numbers. The bleeding times corresponded more closely to the clinical condition, being longest in the two patients who suffered recurrences and lowest in those who had made a complete recovery. One discrepancy will be noted in the case of a child whose symptoms

TABLE I.

Case.	Bleeding Time in Minutes.	Erythrocytes per Cubic Millimetre.	Hæmoglobin Value.	Leucocytes per Cubic Millimetre.	Differential Leucocyte Count—Percentage.					Platelets per Cubic Millimetre.	Splenectomy.	History After Discharge.
					Neutrophile Cells.	Lymphocytes.	Monocytes.	Eosinophile Cells.	Basophile Cells.			
1	3	4,670,000	55	15,000	41	37	10	11	1	4,700	Yes	No bruising even with injury.
2	3½	4,790,000		13,000	45	41	7·5	6		99,000	No	No bruising whatever.
3	6½	4,780,000		10,300	56	27	7	8		49,100	No	Bleeds easily; if smacked, outline of hand appears next day. Epistaxis. Profuse bleeding after tonsillectomy.
4	5	4,280,000		9,300	62	26	9	2·5		53,500	Yes	Can play football without bruising.
5	4	3,990,000		10,600	35	51	5	9		50,700	No	No bruising.
6	3	4,070,000		7,600	67	27	4	1		8,390	No	No bruising.
7	3	3,980,000		10,700	51	40	6	1		[33,170	No	No marked bruising.
8	4½	6,060,000		14,000	77	12	7	2		131,000	No	Constant recurrence of bleeding.

had completely disappeared following splenectomy, and yet the bleeding time remained at five minutes. On the whole, then, the bleeding time appears to give more accurate information than the platelet count.

Anaphylactoid Purpuras.

Anaphylactoid purpuras include: (i) Schönlein's disease or *peliosis rheumatica*, the symptoms of which are stated to be purpura with swelling, pain and tenderness of joints; (ii) Henoch's purpura, which is characterized by abdominal pain, vomiting and melena.

Both of these conditions are, according to the text book descriptions, necessarily accompanied by urticaria. I cannot help feeling that much of the confusion which has arisen in the classification of purpura, derives its origin from the group which is termed "anaphylactoid." Joint pains, abdominal colic and melena are quite frequent symptoms of the common juvenile type of purpura which I have described, and yet many physicians will classify any purpuric condition in which the patient develops joint pains, as *peliosis rheumatica*, or one with abdominal colic and melena as Henoch's purpura. The fact that effusion into a joint takes place or that bleeding occurs from the bowel rather than the nose or kidney, does not appear to be sufficient ground on which to base a separate classification.

The true distinguishing feature of such cases must be the development of purpura in preexisting urticarial wheals, but no instances occurred in this series.

Ætiology of Purpura.

The ætiology of purpura is a very fascinating aspect of the subject. Innumerable theories have been advanced, chief of which are the following: (i) Under-production of platelets by the marrow, as advocated by Frank; (ii) excessive destruction of platelets by the spleen and other parts of the reticulo-endothelial system; (iii) the theory advanced by Duke and advocated by Tidy, that the primary lesion is damage to the capillary endothelium and that the platelets in attempting to patch up their damaged areas are thereby reduced in numbers or completely annihilated, if the damage is extensive.

The chief objection to the first theory is that if the platelets are not produced, then the coagulation time should be prolonged, as they form such an integral part of the blood-clotting mechanism. This, as we have seen, seldom occurs—coagulation time is within normal limits. Furthermore, in the juvenile type of purpura the patient is usually robust and has had no previous tendency to hæmorrhage. It is therefore extremely unlikely that there is any inborn tendency to platelet under-production, but that some new and additional factor must be present.

It is argued in favour of the second theory that the spleen normally destroys effete platelets and that if this function is stimulated by toxic or other influences, the spleen will then attack and destroy all platelets in circulation. It was this conception which suggested to Kaznelson splenectomy as a curative measure, and the results of the operation recorded in the literature have been in many cases highly successful. The chief argument against this theory is that both clinically and experimentally complete absence of platelets may exist without the occurrence of purpura. Numerous instances of this kind have occurred in the literature, and in the series of cases which I have previously detailed, it has been shown that a low platelet count may be associated with a short bleeding time and freedom from hæmorrhagic tendencies.

Bedson experimentally reduced the numbers of platelets in the circulation by the injection of agar serum and purpura did not result; but when anti-red cell serum was also injected into the animal, extensive purpura was produced. Bedson's explanation is that agar serum reduces the number of blood platelets and that anti-red cell serum damages the capillary wall. It is therefore argued that capillary damage as well as platelet destruction is a necessary factor and that an over-active spleen by its action in destroying blood platelets could not be the sole cause of purpura.

The theory that capillary damage is the primary and essential factor offers the most rational explanation and the diminution in the number of the blood platelets must be a closely allied phenomenon, whether it be caused directly by the same agency which plays havoc with the capillary wall, or secondarily as the result of the destruction of blood platelets in their efforts to repair this damage.

Increase in bleeding time and tendency to subcutaneous hæmorrhage after mild traumata certainly point to increased permeability of the capillary wall. The difficult point to determine is the underlying cause of this alteration in capillaries and blood platelets in a previously healthy child.

In many cases of purpura this agency is well known and recognized. In the conditions produced by poisons such as benzol, in the acute infections such as cerebro-spinal meningitis, septicæmia or subacute bacterial endocarditis, it must be a direct toxic effect on the delicate capillary endothelium: in the blood diseases such as leuchæmia and the cachexias of old age, it is probably capillary damage due to defective oxygenation. But when we come to the group commonly termed idiopathic, the difficulty is much greater.

In a certain proportion of the cases I have described purpura followed so closely in the wake of an infection that it may reasonably be assumed that there was a causal relationship between them. For instance, two cases occurred as a complication of pneumonia, another followed a large tooth abscess from the base of which a hæmolytic streptococcus was cultivated, two were preceded by severe facial suppuration and another by a severe attack of measles. A recent *post mortem* examination in a case of influenzal septicæmia at the Royal Alexandra Hospital for Children revealed extensive purpura of both lungs.

The literature also contains many instances of purpura developing as the result of bacterial invasion. Pulvertaft in a recent issue of *The Lancet* records two cases following streptococcal infections of the arm. Bonnel in the *Journal de Médécine de Bordeaux* describes a case of extensive purpura following closely on a purulent conjunctivitis from which a meningococcus was isolated, and meningitis developed at a later date. Other writers describe cases which occurred after the acute exanthemata and in particular after scarlet fever and in fulminating cases a streptococcus has been cultivated from the blood stream.

I have endeavoured to obtain some experimental proof of the ætiology of purpura by the injections of various substances into the ear nerve of rabbits. Purpura is readily produced by the injection of benzol or by feeding the animal with this substance. Injections of peptone into previously sensitized rabbits have given negative results.

Pulvertaft claims that injections of the toxins of hæmolytic streptococci precipitated from broth cultures by alcohol, will produce purpura when injected into a rabbit. Dr. Anderson and, more recently, Dr. Noad have carried out similar experiments for me, and although we have in some cases produced purpuric patches in the lungs and definitely reduced the platelets in the circulation by the injection of the toxins of the *Streptococcus hæmolyticus*, the results were not entirely conclusive.

More recently we have given repeated intravenous injections of the organisms themselves with much more success.

In the case of the *Streptococcus hæmolyticus* intense purpura throughout the lungs and alimentary tract ensued. Similar injections with cultures of the *Bacillus coli communis* gave entirely negative results, and we are now investigating the meningococcus, staphylococcus and pneumococcus.

A considerable amount of evidence can, therefore, be produced to show that in a certain number of cases purpura is produced by bacterial agencies. In others, however, there is no evidence of acute infection, no rise of temperature and no leucocytosis; and these present the chief difficulty.

Large and unhealthy tonsils are almost invariably present and it is possible that, just as in rheumatism, chronic infection in this region and sensitization to a particular organism may account for the condition. A relative increase in the lymphocytes and monocytes which was frequently found in the cases under review, would be quite in accord with this theory.

Anaphylaxis must also be mentioned as a possible cause, but in this series of cases I have seen no evidence of urticaria or other anaphylactoid manifestations to support this theory.

Treatment.

Treatment varies according to the stage and severity of the disease. During the stage of active bleeding in a primary attack, if the condition is mild, rest in bed and protection from trauma are usually sufficient. It is surprising how many of these patients cease bleeding completely without any other treatment.

If the condition is more severe, blood transfusion is essential and usually gives excellent results. Two factors are probably concerned in this—the supply of a number of fresh red blood cells and platelets, and stimulation of the bone marrow. In the fulminating cases treatment is of little avail; death usually occurs in twenty-four to forty-eight hours in spite of transfusion. Splenectomy in such cases is, in my opinion, contraindicated, as the child is too ill to survive an operation.

When the acute stage has passed and the blood platelets remain low, liver extract appears to be beneficial in stimulating their production. Intramuscular injections of milk have been recommended, but I have not been impressed with the results.

The child should remain in bed, if possible, until the bleeding time has shortened and the platelet count increased; a period of three to four weeks is usually necessary.

When the attack has completely subsided, the removal of any septic foci, such as infected teeth or tonsils, should be considered if the blood condition is satisfactory. It is unwise to attempt even the removal of teeth during the early stages. In my enthusiasm to ascertain if a hæmolytic streptococcus was present in an abscess at the root of an infected tooth, I had the tooth removed just after the attack had subsided. I soon regretted the procedure, as the bleeding proved difficult to control. The bleeding time in this case was seven minutes, the coagulation time four minutes, and the platelets too few to count.

In the recurring cases splenectomy usually gives excellent results. In the two cases which I have seen, there has been no recurrence of hæmorrhage and the children are able to indulge in rough sports without ill effects.

This raises the question as to what are the results of splenectomy. They are said to be: (i) An anæmia which is followed by recovery in three or four months, (ii) a leucocytosis reaching as high as 20,000 or even 40,000 per cubic millimetre, (iii) increase in the resistance of the red cells to normal saline solution, (iv) a temporary increase in the number of blood platelets.

Two patients who have for some time past been under the care of Dr. Wade, suffering from a familial jaundice not associated with increase of the red cells, furnished a very interesting example of the results of splenectomy, and Dr. Tidswell has kindly supplied me with detailed figures of the blood counts.

One child, R.C., was subjected to splenectomy by Dr. Wade, and the other, J.C., left *in statu quo*. The blood counts of R.C., who is seven years of age, are set out in Table II. The brother, D.C., was not subjected to splenectomy; his blood counts form an interesting check on his brother's; they are shown in Table III.

TABLE II.

Date.	Erythrocytes per Cubic Millimetre.	Hæmoglobin Value.	Colour Index.	Leucocytes per Cubic Millimetre.	Differential Leucocyte Count—Percentage.				Platelets per Cubic Millimetre.	Fragility: Percentage for Beginning and End of Hæmolysis.
					Polymorphonuclear Cells.	Lymphocytes.	Mononuclear Cells.	Eosinophile Cells.		
24.1.29	1,070,000	19%	0·98	6,300	63	31	4	2	Abundant	
13.2.29	1,100,000	24%	1·09	3,400	72	22	4	2		0·42-0·3
10.4.29[1]	1,300,000	25%	0·90	5,900	74	11	5	8		
17.4.29[2]	2,100,000	35%	0·80	10,500						
22.4.29	2,800,000	32%	0·71	7,700	84	11	4	1		
29.4.29	2,500,000	49%	0·98	7,300	75	16	5	4		
6.5.29	2,940,000	47%	0·59	8,000	74	18	6	2		
14.5.29	2,330,000	50%	1·00	12,200	57	26	11	2		
5.6.29	2,500,000	50%	1·00	12,500	78	13	4	4		
10.6.29	2,000,000	44%	1·10	15,500	73	17	3	7	30,000	
18.6.29	2,570,000	40%	0·78	15,500	74	18	5	3	128,000	
24.6.29	2,320,000	45%	0·98	11,800	73	21	4	2		
3.9.29	2,610,000	40%	0·77	10,400	62	28	6	4	175,000	0·42-0·3
29.5.30	2,780,000	35%	0·65	12,000	60	34	3	2		0·4-0·28
17.6.30	2,340,000	40%	0·80	26,800	65	22	10	3	109,200	0·36-0·28
16.8.30	2,660,000	53%	1·00	22,800	50	30	4	5	450,000	

[1] Transfusion performed.
[2] Splenectomy performed by Dr. Wade.

TABLE III.

Date.	Erythrocytes per Cubic Millimetre.	Hæmoglobin Value.	Colour Index.	Leucocytes per Cubic Millimetre.	Differential Leucocyte Count—Percentage.				Platelets per Cubic Millimetre.	Fragility: Percentage for Beginning and End of Hæmolysis.
					Neutrophile Cells.	Lymphocytes.	Monocytes.	Eosinophile Cells.		
24.1.29	1,820,000	25%	1·00	28,00	72	24	2	2		
13.2.29	1,270,000	22%	0·87	4,300	74	20	4	2	91,600	
8.3.29	1,950,000	20%	1·10	7,200	49	42	2	4		0·4-0·3
11.5.29	860,000	17%	1·00	4,500	80	16	4		49,300	
2.7.29	2,050,000	41%	1·00	4,600	61	30	9			0·44-0·32
17.6.29	2,270,000	35%	0·80	8,400	69	21	10			
16.8.29	2,250,000	80%	0·30	4,800	57	30	10	3	131,000	

Another patient, A.W., under the care of Dr. Wade, suffering from purpura, was subjected to splenectomy and his blood counts are as shown in Table IV.

Unfortunately the resistance of the red cells was not estimated, but the increase in blood platelets soon after the operation was very definite and there was also an increase in the white cell count.

We have endeavoured to estimate the effects of splenectomy on rabbits with the kind surgical assistance of Dr. H. S. Humphries, but unfortunately, although the operative treatment has been successful, the wild rabbits usually succumbed to their unaccustomed treatment before a prolonged study could be made.

One, however, has remained with us and his blood counts are as shown in Table V. Unfortunately a leucocyte count was not done.

All these figures, then, support the results usually accepted as following splenectomy: (i) Definite leucocytosis occurs; (ii) the resistance of the red cells to dilute saline solutions is increased, (iii) the blood platelet count is increased.

It was originally thought, as I have previously stated, that in purpura the spleen destroyed blood platelets in excessive numbers, and that this was the explanation of their increase after splenectomy. The later theory advanced is that the removal of the spleen in some way stimulates the marrow to produce blood platelets.

TABLE IV.

Date.[1]	Erythrocytes per Cubic Millimetre.	Colour Index.	Leucocytes per Cubic Millimetre.	Differential Leucocyte Count—Percentage.				Platelets per Cubic Millimetre.	Coagulation Time.	Bleeding Time.
				Neutrophile Cells.	Lymphocytes.	Monocytes.	Eosinophile Cells.			
5.6.29	4,780,000	71	7,800	77	15	7	1	None found	3 minutes	7 minutes
23.6.29	3,730,000	9	12,800	72	16	8	4	186,500		
21.7.29	4,280,000		9,300	62	26	9	2	52,500		

[1] On June 13, 1929, splenectomy was performed by Dr. Wade.

TABLE V.

Date.[1]	Erythrocytes per Cubic Millimetre.	Platelets per Cubic Millimetre.	Fragility: Percentages at Beginning and End of Hæmolysis.
9.5.30	4,430,000	158,000	0·42 to 0·36
28.5.30	5,370,000	240,000	0·42 to 0·34
30.5.30	4,290,000	218,000	0·42 to 0·32
7.8.30	5,930,000		0·38 to 0·34

[1] Splenectomy was carried out on May 16, 1930.

In this connexion I would again like to quote the case of I.D.

This was a boy, aged four years and four months, who was admitted under the care of Dr. Edgar Stephen, suffering from a severe purpura which occurred just before the crisis of pneumonia. Gangrene of a leg and sloughing of one ear and a large area of the lumbo-sacral region later developed. Amputation of the leg was necessary and the child made a complete recovery.

The blood counts proved to be intensely interesting, although no count was actually made during the period of active bleeding. They are set out in Table VI.

The comparison of the platelet count and that of A.W. following splenectomy is interesting. In the latter splenectomy was followed in ten days by an increase in the blood platelets from nil to 186,500. In the case of I.D. considerable sloughing of tissues and the small operation of merely severing the gangrenous part of the leg, were followed in nineteen days by an increase in the platelet count from 150,000 to 539,000. The later history of both children shows that they have been completely free from purpura and are able to withstand hard knocks without undue bruising.

It is a well recognized fact that the platelet count increases after operations and confinements and that thrombosis may follow as a result.

The benefit derived in purpura from blood transfusion may not be due entirely to the introduction of a certain number of fresh blood cells and platelets, but also to marrow stimulation by the products of these cells.

Has splenectomy, then, a purely specific action in increasing the numbers of blood platelets, or may this result not be due in part at least to the breaking down of cells by operative procedures, just as we have seen in the case of the boy who suffered from sloughing and gangrene? The question is a very interesting one and may have a direct bearing on treatment, because if there were any truth in this hypothesis, the administration of some form of tissue extracts would be well worthy of a trial.

Acknowledgements.

In conclusion I wish to express my thanks to the senior physicians of the Royal Alexandra Hospital for Children for their kindness in allowing me access to their patients and their histories, and in particular to Dr. Tidswell and his assistants in the Pathological Department of the Hospital, Dr. Anderson, Dr. Noad and Dr. Bradley, for their untiring assistance in carrying out the technical investigations.

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TABLE VI.

Date.[1]	Erythrocytes per Cubic Millimetre.	Leucocytes per Cubic Millimetre.	Differential Leucocyte Count—Percentage.			Platelets per Cubic Millimetre.
			Neutrophile Cells.	Lymphocytes.	Monocytes.	
28.10.27	2,125,000	40,000	87	8	5	206,500
11.11.27	2,380,000	13,000	55	35	9	50,858
24.11.27[2]	3,510,000	10,000	53	43	2	150,000
13.12.27	4,150,000	7,200	50	27	20	539,000
22.7.30	4,790,000	13,000	45	41	7·5	99,000

[1] On October 23, 1927, the patient was admitted with intense purpura; a crisis of pneumonia had just occurred. On October 25, 1927, blood transfusion was given; no fresh hæmorrhages had taken place.
[2] On November 24, 1927, the foot was amputated.

OBSERVATIONS ON THE ACTION OF COLLOIDAL LEAD ON AN IN VITRO CULTURE WITH REFERENCE TO SUPPLEMENTARY IRRADIATION.[1]

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BLAIR BELL proposed the use of lead preparations for the treatment of malignant disease on the analogy that lead salts caused abortion through a specifically toxic action on the chorionic cells.(1) It is probable that such action applies to any cells of any embryonic or partially dedifferentiated character. Of various preparations tried, a colloidal suspension of metallic lead proved to be least toxic to the body tissues generally, whilst showing some favourable results in the case of cancerous tissues. Attempts to reinforce the action of colloidal lead by means of X or γ radiation have been disappointing, the results being less favourable than with either agent acting alone. In the present work I endeavoured to obtain further information from the behaviour of the dedifferentiated cells of an *in vitro* culture when it was subjected to the action of colloidal lead preparations with or without X or γ radiation.

Method of Culture.

A fragment of heart muscle from an eight day chick embryo was cultured in a hanging drop of amniotic fluid following a technique described in an earlier paper.(2)

Brass plates with a circular aperture closed by cover slips were used in place of cavity slides to facilitate observation and X ray treatment. A very good growth is obtained after twenty-four hours' incubation, many of the cells being obviously fibroblasts, whilst other cells which tend to give a carpet-like growth, are probably dedifferentiated muscle cells. Toward the end of the second day degeneration sets in, the amniotic fluid serving as a physiologically neutral solution rather than a true growth medium, but ample time is available for an investigation of this kind.

Lead Preparation Used.

The colloidal lead was prepared by Mr. Newman, who followed approximately Blair Bell's technique, except that the average size of the particles was increased by centrifuging and drawing off a particular layer in order to facilitate observation. Such preparations show metallic particles in free Brownian movement, varying in size from about 0·3 μ to the limits of resolution. The colloidal character of the particles appears to depend on some special condition of the surface and with access of oxygen they become aggregated and fall out of suspension, later becoming converted into a white hydroxide. Even in the fresh state such preparations contain about 25% lead hydroxide and 10% lead carbonate together with some gelatin to aid suspension. In some experiments a colloidal lead phosphate was used as indicated in the tabulated results, but the extreme fineness of the particles rendered observation difficult, although the preparation has the advantage of stability. The lead sol was transferred to the cultures by means of a small glass rod about one millimetre in diameter, which carried a quantity about one-twentieth of the volume of the drop of amniotic fluid.

Identification of Ingested Particles.

Dilling has described the following tests for the identification of ingested lead particles.(3)

1. They are soluble in glacial acetic acid and 50% nitric acid, insoluble in 10% acetic and nitric acids.
2. They appear blacker with ammonium sulphide.
3. They become yellow with potassium iodide and dissolve in excess.
4. They become an orange-red with chromates.

Reference was also made to the fact that tests for other metals such as iron should give no reaction. In the present work I relied entirely on the formation of lead chromate, adequate controls always being carried out. After thirty hours' incubation the specimens were fixed and stained according to the following technique. They were first washed with saline solution in an inverted position in order to remove the excess of lead particles or other "floating" impurities. Saturated potassium chromate was then run in to act as a fixing agent and to stain lead particles an orange-red. After washing the preparations were counterstained with methylene blue and mounted in balsam in the usual manner.

Experimental Technique and Observations.

The experiments which are recorded in Table I were carried out in batches of six, the slides being labelled A, B, C, D, E, F for convenience. A, B, C and D received a dose of colloidal lead and A and B were also subjected to radiation treatment, whilst C and D served as a comparison. Slides E and F did not receive any colloidal lead, but E received the same radiation treatment as A and B of the same series. Of the 150 specimens set up, 63 were lost owing to poor growth, infection, floating off during fixation *et cetera,* as indicated by the blank spaces in the table.

In recording the successful experiments N represents a perfectly normal appearance (Figure I) and S a slight degree of degeneration which one associated with the ageing culture.

The toxic lead sol, which was usually added when setting up the culture, caused definite degenerative changes (represented by D), although sufficient growth took place to permit of observation. The carpet-like cells showed extreme changes and observation was confined to the fibroblasts which showed a ragged outline and shrinkage and displacement of the nucleus. Fatty degeneration was not observed. Ingested particles of lead were readily identified when large, but the majority were on the

[1] This work was carried out under the control of the Cancer Research Committee of the University of Sydney and with the aid of the Cancer Research and Treatment Fund.

TABLE I.

Observation:	Five Minutes Mixed Rays.				Ten Minutes Mixed Rays.					Five Minutes Mixed Rays.						Thirty Hours Radium D.								
	1	2	α 3	4	5	β 6	β 7	γ 8	9	δ 10	δ 11	12	13	δ 14	δ 15	16	17	α 18	19	20	21	22	23	24	25
A Lead and Radiation ..	D	D	S	D			D	S	D			D				D+		D+	D+	D+	D+	D+	D+	D+	D+
B Lead and Radiation ..	D		S	D			D	S	D			D						D+	D+	D+	D+	D+	D+	D+	D+
C Lead	D		S	D		S		S	D			D							D	D	D	D	D	D	D
D Lead	D		S	D		S	D		D							D			D	D	D	D	D	D	D
E Control	N	N	N			N	S					N	N			N		N	N		S	N	N	N	S
F Control	N	N	N	N			S	N				N	N						N	N		S	S	S	N

N = normal.
S = slight degeneration.
D = moderate degeneration.
D+ = extreme degeneration.

α = lead phosphate.
β = lead irradiated separately.
γ = lead added after twenty hours' growth.
δ = infection.

limits of resolution and often caused a purple discoloration of parts of the cell rather than discrete reddish points. In some cases vacuoles were observed round the lead particles and the distribution was often perinuclear. A fairly successful colour representation is shown in Figure II, which may be compared with the normal cells in Figure I, photographed in a similar manner.

Results.

Turning now to the tabulated results, the addition of lead always caused degeneration. This was not appreciably affected when the specimen was exposed for five to ten minutes to unfiltered mixed X rays, 30 centimetres from a water-cooled Coolidge tube running at 50 kilovolts and 10 milliampères (Series 1 to 15). As indicated by the small changes (S) lead phosphate proved unsuitable, although similar in action, and the addition of lead after twenty hours, when growth was established, did not give such good results. The controls E and F showed that the small dose of radiation did not produce any visible change, since slight changes associated with age appeared indiscriminately.

Experiments with γ Radiation.

In the remaining series, 16 to 25, radiation was supplied by a specimen of radium D, for which I am indebted to Professor Briggs. A homogeneous band of γ rays is given out corresponding to 46 kilovolts or 0·27 Ångström unit approximately, which is much softer than the hard γ rays from radium B and C. The intensity was comparatively feeble, approximately equivalent to one milligramme of radium, and the specimens were exposed for thirty hours to give a dose of the same magnitude as the mixed X rays used previously. Slides A, B and E were placed in a cavity on a lead block adjacent to the radium D with a thin lead plate intervening to screen off β radiation, the whole being placed in an incubator. The control E was placed nearest to the radium tube, but in accordance with the previous experiments it showed no change attributable to radiation. The specimens containing lead showed much greater degenerative changes, represented by D+, when exposed to radium D. Ingestion of lead also appeared to be increased and a coloured microphotograph of such a specimen is shown in Figure III. The red granules are somewhat exaggerated and the background and dust particles show reddish in all figures, but the purple of the cytoplasm is indicated and in the main the three figures give an accurate comparison of the changes to be observed.

Discussion.

It has been shown that the γ rays from radium D reinforce the destructive action of colloidal lead, whilst mixed X rays are without effect.

As the doses were of similar magnitude, the difference in action may be due to a time effect (the distribution of the radiation over thirty hours as against five or ten minutes) or to some specific selective action of the radiation employed. As mentioned above, radium D gives a homogeneous radiation, but it is quite probable that the easily absorbed secondary radiation from the lead walls of the cavity was mainly responsible for the effect.

It is also doubtful whether the action of the rays is on the lead, the cell or both. Probably the atoms of the colloidal lead become ionized and pass more readily through the cell membrane, since the cells appear to be unaffected by the radiation alone. Irradiation of the lead separately was tried (see Table I), but such an experiment is likely to be inconclusive, since the atoms would presumably return to a normal state in a short time.

The experiments suggest that one might reinforce the action of colloidal lead preparations on cancerous tissue by a local application of radiation for about twenty-four hours after injection or as long as the lead circulates in the blood. Ideally one would use a feeble source of radium D, which is the end

product, in old emanation tubes, and screen the rays through an extremely thin lamina of lead in imitation of the above experimental conditions.

I regret that Dr. Cardamatis was unable to share in this work, as intended, but we are carrying out clinical investigations on the lines indicated.

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POSTOPERATIVE ABDOMINAL ADHESIONS.

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POSTOPERATIVE adhesions are perhaps not an attractive subject to the surgeon who, when a patient complains of pain at the site of the operation area, cheerily advises him to forget it and not to let it worry him. This is truly easy advice to give, but it is founded on a very prevalent false idea that the pain is not due to any organic lesion, but to a functional cause.

A long and wide experience of abdominal surgery, together with that close observation of patients which is necessary in a teaching appointment at a public hospital, has convinced me that we shut our eyes to facts when we class a majority of these cases as functional. Having blamed the patient for worrying about his or her trouble and having ascribed the pain to an unstable nervous system, we proceed to give nerve sedatives, such as bromides, and advise the patients not to become introspective about their trouble. Sometimes they are classified under that exceedingly elastic and much abused term of neurasthenia. We do them an injustice, in fact, many.

The earliest consists of perhaps that not too careful handling of the abdominal contents and of the parietal peritoneum at the first operation; this has caused an exudate of lymph on the delicate endothelial surface of the bowel, omentum and peritoneum; these roughened surfaces become adherent to one another; the resultant traction upon the parietal peritoneum (well endowed with sensory nerves) causes the pain. Adhesion of visceral peritoneum to visceral peritoneum may be quite symptomless if there is no mechanical obstruction to the alimentary canal.

At the site of the adhesions to the parietal peritoneum there is—constantly in my experience—marked hyperæmia—further cause for the pain that is felt.

Why are some cases followed by adhesions and others not? I do not know.

There are several contributing factors:

1. Trauma, because of rough handling and too free evisceration. Attempting to work through too small an incision is frequently a mistake; it favours rough surgery.

2. I have an idea that the too free use of retractors is sometimes a factor. I am quite well aware of their advantages in giving a good exposure, but parietal peritoneum needs very gentle handling. I find that strong forceps, like Moynihan's or Lane's, gripping the edges of the incision give one most of the traction one wants.

3. Naturally, the insertion of a drainage tube encourages the formation of adhesions, but one cannot abandon what is in many cases a life-saving measure because of possible after-pain.

4. The fact remains that in many cases adhesions follow unexpectedly after some operations and do not in others where we might reasonably have expected them. There is an unknown factor.

Is it the pathogenicity of the intestinal flora, superadded to slight trauma, which results in their appearance sometimes? I do not know.

Symptoms and Signs.

The pain is more or less constant, but worse on movement; it is generally independent of meals. This fact may be helpful when the scars are in the epigastric region, for the pain might be due to a recurrence of the gastric or duodenal ulcer trouble, for which he had been originally operated upon, and not to adhesions. A barium meal will be helpful.

In some cases it may not come on for several years after operation (presumably the adhesions are cicatrizing and thus slow in exerting their pull), but this is not usual. There may be occasionally some difficulty in getting the bowels to move, due to adhesions partially obstructing the bowel.

The presence of one or more scars on the abdomen should always suggest the possibility of adhesions. Invariably there is tenderness on palpation over the scar, especially over that part where the adhesions are and where the hyperæmia is most marked. There are no other tests of any value that I am aware of, except in excluding other organic diseases.

That they suggest functional disturbance of the nervous system is perhaps not surprising in view of the vague nature of the signs and symptoms and the wound to his pride by telling a surgeon that there is still pain at the site of an operation scar. What is more natural than to say that it is functional when the appendix or other organ causing the original trouble has been removed? The surgeon seems to consider it due to his self-respect to call it functional.

Treatment.

These patients are not popular and yet they are deserving of a great deal of consideration. In some cases they can be quite cured, the remainder much

relieved. The amount of relief depends upon the extent of the adhesions; if over a wide area, it is only partial; if more localized, usually complete.

Many methods have been used in days gone by for their relief, such as the introduction of olive oil, liquid paraffin and other substances into the peritoneal cavity after division of the adhesions; also the covering of the raw area with Cargile membrane. These I have abandoned and, having divided the adhesions and removed the redundant portions, proceed to cover over the raw areas, whether of bowel, omentum or parietal peritoneum, by bringing together the edges of the healthy peritoneum, thus inverting and covering in the roughened areas. This, in the case of the parietal peritoneum, may prove no easy matter. A capable assistant is always helpful. A Reverdin needle may be of great value and strong catgut (plain or chromicized) sutures are advisable (the strain upon them is often great).

The rough area is sometimes too wide to be covered in completely and one must be satisfied with a partial operation and partial cure. Later, if considered advisable, a further operation may be done to complete it.

As to be expected, there is generally a considerable amount of pain for several days.

I have in some cases supplemented the operation by irrigation of the colon with saline solution for several weeks. Whether this is of value is, of course, debatable; it is not an essential.

I could quote a large number of clinical cases in support of my contention that the operation I have outlined is of great value, but they tell much the same story. I have practically summarized them in this article.

Reports of Cases.

A SEQUEL OF VESICAL CALCULUS.[1]

By W. E. HARRISON, M.B., B.S. (Melbourne),
Honorary Surgeon to Out-Patients, Base Hospital, Bendigo, Victoria.

ON December 2, 1929, after returning from a day out shooting, the patient, a male, aged sixty-five years, felt chilly with generalized aches and pains. The next morning, when I first saw him, his temperature was 39·1° C. (102·5° F.); he had a rigor before I left. The urine that afternoon showed nothing abnormal on chemical and microscopical examination. He gave a history of a Neisserian infection about thirty years previously.

The next day, two days after the onset of his illness, he had difficulty in passing urine and drops of blood were coming from the urethra; there was tenderness in the position of the bulb of the urethra and slightly over the prostate, but no obvious swelling could be made out. On the following day, the third day of his illness, the penis commenced to show signs of gangrene and became intensely swollen and bent on itself in much the same way as one sees in infiltration of the *corpus spongiosum* from acute inflammation. The scrotum was greatly swollen and indurated and with the skin over the hypogastric and inguinal regions changed to a dusky colour. No fluctuation could be made out anywhere. By this time there was complete retention of urine. Dr. Long and Dr. Little saw the patient with me and a provisional diagnosis of a periurethral extravasation of blood, possibly the result of a periuretaral abscess, was made.

A suprapubic cystotomy was done under local anæsthesia. At first what appeared to be normal urine came away, but on feeling around the base of the bladder a quantity of old blood clot and pus welled up. A De Pezzer catheter was left in the suprapubic opening and attached to a suction apparatus.

In a few days the induration and swelling of the penis and scrotum began to subside and two openings spontaneously formed, one under the scrotum in the mid-line and another at the junction of the dorsum of the penis with the pubis. These were enlarged and through them old blood clot and pus discharged, similar to that found in the base of the bladder. At times the discharge through the opening on the dorsum of the penis had a fæcal odour and the patient felt sure that flatus was coming through it. In a couple of weeks the gangrenous part of the penis, which involved only the skin and subcutaneous tissues of the dorsum and to a lesser extent the lateral surfaces, separated.

Six weeks from the onset of the trouble a rubber catheter, when introduced into the urethra, was found to enter the rectum about 2·5 centimetres (one inch) above the anal sphincter through an opening big enough to admit the tip of a finger. The same thing happened on introduction of a medium size sound. Obviously, therefore, he had a recto-urethral fistula. This healed in another four weeks and a number 9 metal catheter was introduced into the urethra and entered the bladder without difficulty. The catheter was tied in and the De Pezzer catheter removed and the suprapubic opening allowed to close. The metal catheter was finally left out in another five weeks (during this time it was occasionally replaced by a rubber one) and to our pleasant surprise he passed urine naturally and has been able to do so since and has no incontinence of urine.

Before he left hospital, about a month ago, all the external parts had healed, except a small opening under the scrotum in the mid-line. No methylene blue, when given orally, was observed to come through this opening, but now it is quite obvious that a little urine comes through it on micturition. Four days ago I found that I could not pass even a small size sound into the bladder; the tip was arrested by something hard and grating within 2·5 centimetres (an inch) of the triangular ligament. As I was uncertain as to whether a calculus was present or a hard fibrous stricture was forming, a skiagram was taken (unfortunately without the catheter left *in situ*) and this shows a shadow in the line of the urethra. A probe, when passed into the small opening in the scrotum, goes in for about 7·5 centimetres (three inches) to reach the position of the tip of the sound in the region of which a hard, movable body about the size of a small cherry can be felt.

On June 6, a fortnight subsequent to showing the patient at our Bendigo meeting, a calculus which presented at the scrotal sinus, was removed. The calculus which has not been chemically examined (it is possible, but very unlikely, that it has formed since the trouble began) measured 18 by 12 millimetres (three-quarters by half an inch). Only on micturition urine comes through the opening, but in larger quantity than before the calculus was removed. The patient is prepared for the present to put up with the discomfort of a urethral fistula.

Comments.

The discovery of a calculus offered an explanation of what at first seemed to be a very rare condition. Apparently a vesical calculus in an attempt to be passed *per urethram* ulcerated through the walls of the urethra and surrounding vascular tissues and septic contamination by urine supervened.

Although it is common enough for calculi to be passed *per urethram*, the amount of tissue damage and pain depending on the size and shape of the calculi and calibre of the urethra, there are several features associated with the history of events in this patient which I cannot find described on the subject:

[1] The patient described herein was shown at a meeting of the Victorian Branch of the British Medical Association, Bendigo, May 24, 1930.

1. The large amount of extravasated blood and apparently little, if any, of extravasated urine.

2. The comparative absence of pain as distinguished from difficult micturition in the first two days of his illness.

3. The sudden onset of gangrene and really what threatened to be an inevitable widespread destruction of genital organs.

4. The serious damage to the local parts and even the danger to life for which a relatively small vesical calculus may be responsible if left untreated.

5. The urgency of an early investigation (which we failed to carry out) for the detection of a calculus in a patient with hæmaturia and difficult micturition.

Reviews.

LABORATORY METHODS.

"CHEMICAL METHODS IN CLINICAL MEDICINE," by Dr. G. A. Harrison admirably justifies its chosen subtitle: "Their application and interpretation with the technique of the simple tests."[1] Few volumes keep so strictly to the selected avenues of discussion. In only one place does the author's enthusiasm cause a digression and that is surely a pardonable one. In discussing renal efficiency tests he details the microscopical urinary findings. The volume bears the unmistakable stamp of the practical laboratory worker and the sound clinical observer. Hence the technique outlined is practically faultless and the interpretation of results equally valuable. The book comprises twenty-six chapters and a glance at the headings alone arouses an appetite for further delving.

In "The Routine Chemical Examination of the Urine" through "Tests of Renal Efficiency," "The Interpretation of Blood Sugar Curves," "Blood Analysis," "Gastric Analysis" and so on, few methods in clinical chemical pathology remain untreated.

Technical methods are given with meticulous accuracy. The reader's familiarity with calculations *et cetera* is not taken for granted. Dr. Harrison gives these in detail and for the student such a method is of incalculable benefit.

More and more is pathological investigation being found essential for accurate diagnosis and scientifically controlled treatment. As an example, who nowadays would treat a patient for glycosuria without a preliminary glucose tolerance test? Sir Arthur Keith in a recent lecture said: "The initiative in medicine has passed, or is passing, from the clinician into the hands of the laboratory worker," and the majority hold the same view. The absolute necessity for every medical practitioner and medical student to appreciate the valuable aid of pathological investigation in the rational treatment of disease renders this book practically indispensable. Dr. Harrison strikes the right note when he urges closer collaboration between the laboratory worker and the physician.

Chemical and other pathological tests are aids to diagnosis and no sane man delivers a verdict until all facts are placed before him. Hence, in addition to clinical acumen, close observation and common sense, the modern physician demands laboratory facts. This book should be read and digested by every man practising medicine, since it acts the part of a "liaison officer" between the physician and the laboratory.

We have said little of the technical methods supplied for the various tests simply because we can merely endorse them as the best. In the estimation of the glucose content of the blood Dr. Harrison prefers the method of Hagedorn and Norman-Jensen. As a means of improving the stability of the iodide-sulphate-chloride solution he advocates the addition of one gramme of potassium iodide to forty cubic centimetres of the other ingredients for use as required. This we have found to be unnecessary, as the addition of chemically pure metallic zinc prevents complete oxidation of the iodide to iodine. Thus the whole iodide-sulphate-chloride solution may be made up at once and lasts indefinitely.

The various methods of gastric analysis are given in detail and again we must agree with his preference for the fractional method in which samples are withdrawn every fifteen minutes until the stomach is empty. This takes "two or three hours" and every fractional test meal should be carried on for three hours for accurate results and to explore the mine of information available.

The chapter on basal metabolism is complete as regards the evaluation of the clinical value of such tests, preparation of the patient *et cetera*, but we regret to note the only technique outlined is that with the aid of the Benedict portable metabolism apparatus, "since it is the only type of method likely to be used by the practitioner." The method elaborated by Wardlaw using Douglas bags and subsequent absorption of expired carbon dioxide by soda lime should have a place in every modern publication on this subject.

The final chapter on miscellanea and conundrums will surely raise a reminiscent smile on the face of every practical laboratory worker. The self same problems have been presented to us and others in addition. A very comprehensive index completes this valuable work. Finally, we have no hesitation in urging every medical practitioner, and every medical student particularly, to read and keep this book—unique in its way, being technical, yet enjoyably readable.

[1] "Chemical Methods in Clinical Medicine, their Application and Interpretation with the Technique of the Simple Tests," by G. A. Harrison, B.A., M.D., B.Ch., M.R.C.S., L.R.C.P.; 1930. London: J. and A. Churchill. Royal 8vo., pp. 544, with two colour plates and sixty-three illustrations. Price: 18s. net.

Notes on Books, Current Journals and New Appliances.

"THE REVIEW," ADELAIDE.

THE REVIEW, "the journal of the Adelaide Medical Students' Society," has been presented in new garb. In its old form it had a long and useful career. It has appeared now in crown quarto size with an attractive blue cover. The general style of the contents of the journal has not altered; if anything, it is a little more respectable. Graduates of the University of Adelaide are advised that they may obtain *The Review* by becoming graduate members of the Adelaide Medical Students' Society. The subscription to the society is five shillings *per annum*. Three guineas will secure life membership. Graduates should not need urging to join their own society.

PATERNITY.

"To fathers, those tired patient souls, criticized by maternal kin, humiliated by head nurses, ignored by doctors. To those unsung heroes who receive the blame without the glory, but who, at every turn, and after every baby, must pay," Douglas Vass Martin, Junior, has dedicated "Expectant Fathers, Their Care and Treatment." This is an amusing little book which certainly presents a new point of view.[1]

There is a preface "for doctors and nurses only"—"In spite of the vast progress in the realms of gynæcology, obstetrics and pædiatrics, little thought has been given to the problem of ameliorating the condition of the father." The purpose of the book is "not, of course, to detract from the glory of womanhood, but to bring man, the father, to that lofty position which his genius merits." There are sections on "Expectancy Defined," "Symptoms," "The Waiting Period," "Sojourn at the Hospital," "Some Rules to be Observed," "The Economic Aspect of Expectancy," and so forth. The illustrations are wonderful. No medical practitioner will fail to find in this book something to stimulate his amusement centre.

[1] "Expectant Fathers, their Care and Treatment," by Douglas Vass Martin, Junior, with illustrations by R. Wilson McCoy; 1930. Australia: Angus and Robertson Limited. Crown 8vo., pp. 50.

ILLUSTRATIONS TO THE ARTICLE BY DR. W. MOPPETT.

FIGURE I.
Showing normal fibroblasts stained with methylene blue.

FIGURE II.
Showing degeneration caused by colloidal lead. Intracellular particles of lead chromate may be seen.

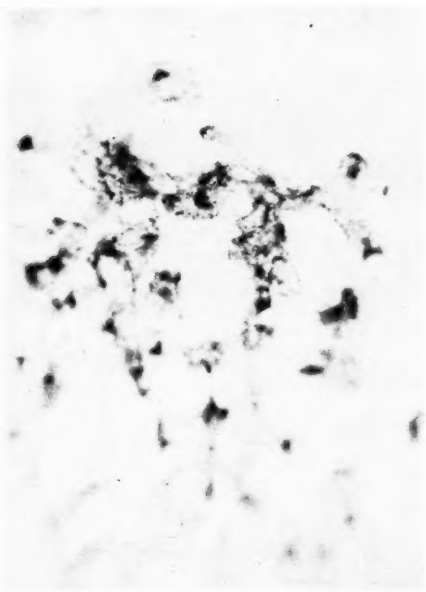

FIGURE III.
Showing extreme degenerative changes caused by colloidal lead and radiation treatment. Intracellular particles of lead chromate may be seen and the methylene blue is rendered a dark purple owing to the presence of ultramicroscopic particles.

NEVILLE REGINALD HOWSE.

The Medical Journal of Australia

SATURDAY, NOVEMBER 22, 1930.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

THE APPENDICITIS PROBLEM.

So much is known about appendicitis, so much has been written about it, and medical practitioners in Australia are so accustomed to its clinical manifestations, that it would appear at first sight as though nothing new or noteworthy could be said about it. Even if there be nothing new to bring forward, the fact that it occurs so frequently and that Australian medical practitioners situated at a distance from large surgical centres are often compelled to undertake appendicectomy would justify a critical consideration of both the diagnosis and treatment. Added to this there is the tendency among some medical practitioners to regard appendicitis as a common complaint and appendicectomy as an operation as simple as shelling peas. It is not necessary to look for something new. Progress has certainly been made, but there still remains a high mortality—a mortality that unfortunately is taken almost for granted. Of course, there occur cases of appendicitis so fulminant that the patient dies in spite of all that may be done for him. Apart from these infections, however, it must be recognized that in the majority of fatal cases someone has blundered. The patient may have treated his illness lightly or he may have treated himself to aperients that open not only his bowels, but an inflamed and weakened area in the appendiceal wall as well, giving rise to general peritonitis. Perhaps the medical attendant has made a mistaken diagnosis, perhaps he has been guilty of the heinous crime of ordering an aperient, perhaps he has failed to realize that immediate operation is the only safe method of treatment and has waited for the attack to subside.

Attention has recently been directed to the mortality of appendicitis by Dr. C. Jeff Miller, of New Orleans, United States of America, in *The Journal of the College of Surgeons of Australasia*. He expresses his views thus:

> Advances in medical knowledge, refinements in diagnosis and technique, elaboration of equipment, have gotten us nowhere. Appendicitis still achieves a mortality which makes it a menace to humanity, a disgrace to the medical profession, a challenge to us who claim to practise the science and art of surgery.

Dr. Miller has investigated the death rate of acute appendicitis for the last six years at two of the largest hospitals in New Orleans. He finds that of 2,415 patients 239 or 9·9% died. This must be regarded as too high a death rate for a condition that is so well understood as appendicitis. New Orleans is a large centre; it has within its boundaries competent surgeons and in this way may be compared to any of the large centres in Australia.

The reduction of mortality from appendicitis will depend on early diagnosis and on efficient treatment. The diagnosis is discussed elsewhere in this issue by Dr. J. Ramsay, of Launceston. Medical practitioners are advised to read carefully what Dr. Ramsay has written, for he has covered the ground in a complete fashion. Although familiar with the vagaries of the appendix, they will probably find food for thought in the facts set out and the method of their presentation. The question of treatment includes what the medical attendant orders and what the patient does for himself. Most medical practitioners realize that patients do irreparable harm to themselves by taking aperients when the appendix is the seat of an acute inflammation, and hospital surgeons know that occasionally medical practitioners are guilty of the offence of ordering aperients (it is not necessary to quote Dr. Miller's figures dealing with this point). For the medical

practitioner there is no excuse, for the patient there is some. While medical practitioners do this thing, it is hopeless to expect the patient to receive teaching, for the only person who can teach the patient is the general practitioner. The final question is that of operation, and here it cannot be too strongly emphasized that surgical removal of the appendix, as soon as the diagnosis is made, is the only thing to be considered. Some physicians may and probably will object to this statement, as they have done in the past. They may be able to point to many patients whose attacks have subsided, but without question more lives would be saved if the habit of immediate operation were universally adopted.

Current Comment.

TRACHOMA.

TRACHOMA has been known and recognized for many centuries. It has been the source of immeasurable suffering, and people in untold numbers have been condemned by it to the gloom and misery of blindness. At various times it has swept like a plague across the countryside in those lands cursed by its presence. The soldiers of the Napoleonic wars suffered in great numbers from trachoma, and they scattered it through Europe. In 1840, when every trachomatous soldier was discharged from the Belgian Army, Belgium became a veritable hotbed of infection.

A century ago acute trachoma was a common and virulent disease, but in this form it is not so frequently seen nowadays, and its virulence has diminished. The incidence is lower, yet in Egypt it is said that a native with normal lids is a rarity. In India, in all tropical countries, and here in Australia the disease is prevalent; all over the world it has been studied clinically and in the laboratory, yet its ætiology is still undetermined and still it sometimes casts before its victims the sombre threatening shadow of impending blindness.

Though most authorities agree as to the infectious nature of trachoma, even this has been disputed from time to time, and there is considerable divergence of opinion concerning the degree of infectivity of a person suffering from the disease. Many authorities believe that some factor in addition to infection is concerned, for example, vitamin deficiency. Harvey Sutton in 1928 suggested that the decline in the incidence of trachoma among school children in western New South Wales may have been due to the increase in wheat cultivation, with consequent closer settlement, improved living conditions and better food supply. Against this theory it might be pointed out that improved facilities for obtaining treatment, and education in prophylaxis, together with improved water supply and hygiene, may have been the responsible factors.

The discovery of the inclusion bodies of Prowazek and Halberstaedter at first gave rise to hope that the revelation of the cause of trachoma might be at hand, but these bodies have since been observed in the vagina, in so-called inclusion conjunctivitis and in follicular conjunctivitis of monkeys. Noguchi and Cohen even went so far as to say that the Prowazek-Halberstaedter bodies could only be found in trachomatous lesions when inclusion conjunctivitis existed as a complication.

When in 1928 Noguchi described the *Bacterium granulosis* and its effects when inoculated into the lids of *rhesus* monkeys it was thought that at long last the ætiology of trachoma had been determined. But Noguchi's experiments have been repeated to the satisfaction of only one or two observers. Furthermore, Lindner, of Vienna, declared that the lesions produced in monkeys inoculated with *Bacterium granulosis* were not trachomatous, and Mayou, of England, expressed the opinion that the histological appearances were not those of trachoma as seen in England. Noguchi's experiments appear to lose some of their value when it is considered that monkeys are liable to a follicular conjunctivitis which may simulate trachoma.

An interesting and useful review of the present state of knowledge of the ætiology of trachoma has recently been presented by Charles Weiss, of the Department of Ophthalmology, Washington University School of Medicine.[1]

Weiss states that the ætiology of trachoma will probably not be understood until the following six questions have been answered: (i) Is trachoma primarily a disease of dietary deficiency? (ii) Is it a local ocular manifestation of a systemic infection or metabolic derangement? (iii) Is trachoma the result of a single specific infection or is it due to a series of infections, possibly of similar or of dissimilar character? (iv) Is trachoma a transmissible disease? (v) Is it due to a filtrable virus? (vi) Is it due to a protozoan parasite or other specific microorganism?

In discussing the first question he points out that at least one disease of the eye, namely xerophthalmia, is due to vitamin deficiency. He states, however, that there is no epidemiological or experimental evidence sufficient to confirm the belief that trachoma is essentially a deficiency disease. On the other hand, it has been shown by Goldberger that the administration of vitamins has no effect on the progress of the disease, and several observers have noted the occurrence of trachoma among well nourished people. Nevertheless, Weiss points out that investigation has yet to be made into the possibility that food deficiency may play a part in rendering the mucous membrane of the eye susceptible to a specific infective agency.

[1] *The Journal of Infectious Diseases*, August, 1930.

He is unable to answer the second question, but admits the possibility that the answer eventually provided may be in the affirmative. The third question provides a problem which may perhaps be solved by the cooperation of clinicians and bacteriologists. A great deal of careful work would have to be done, and many diseases of the conjunctiva would have to be observed from their initial stages to cure or the formation of scar tissue. Study would be necessary of such conditions as folliculosis, follicular conjunctivitis, papillary conjunctivitis, chronic blenorrhœa, vernal catarrh, Parinaud's conjunctivitis, tuberculosis of the conjunctiva, and conjunctivitis due to foreign bodies, sporotrichosis and syphilis.

In reply to the fourth question he remarks that trachoma is most likely a transmissible disease and that support is lent to this view by its geographical distribution, its occurrence in epidemics and its history. It has been said that trachoma was spread by the Mohammedans through the lands which they had invaded for the purpose of spreading the teachings of the Prophet. The conquering Spaniards carried the disease to America. And so trachoma has been disseminated until today it is a serious problem of public health administration throughout the world. Furthermore, various workers claim to have transmitted the disease experimentally from man to man. It is worthy of note, however, that at least one observer has suggested that inoculation merely introduces secondary infection into an eye already affected with a trachomatous or pretrachomatous condition. As Weiss points out, the belief that trachoma is a specific infectious disease can be confirmed only by the discovery of a specific organism conforming to Koch's postulates. It is unfortunate that none of the common laboratory animals are susceptible to trachoma. The reports of the successful experimental inoculation of trachoma into monkeys should not be unreservedly accepted, in view of the liability of these animals to contract so-called spontaneous granular conjunctivitis.

Evidence is conflicting regarding the possible existence of a filtrable virus as a factor in the causation of trachoma. Recent researches into the mechanism of filtration have demonstrated the existence of many factors which may influence the result of a filtration experiment. Further investigation of this phase of the problem is necessary.

The sixth question is one which, since 1881, numbers of investigators have vainly endeavoured to answer. Numerous cocci, yeasts and fungi, the Koch-Weeks bacillus, possibly Pfeiffer's bacillus, bodies resembling protozoa but since shown to be artefacts, *et cetera*, have all from time to time been suggested as the cause of trachoma. Then came the discovery of the inclusion bodies of Prowazek and Halberstaedter. Weiss deals fully with the history of these bodies and proceeds to a discussion of Noguchi's work. There is a diversity of opinion concerning this, and it may be taken that much work is required before Noguchi's findings can be accepted or discarded. Weiss points out that the follicular conjunctivitis of monkeys may actually correspond to trachoma of man and that it may be impossible for the typical lesions of trachoma to appear in monkeys only on account of the poor development of lymphadenoid tissue in these animals. Studies in immunity, search for the presence of antibodies to *Bacterium granulosis* and search for *Bacterium granulosis* in all stages of trachoma are among the more important investigations which are necessary. Weiss is unable to answer one of the six questions he has put, but rather asks in turn many more. Though the natural impulse is eagerly to welcome Noguchi's discovery, maturer thought breeds an attitude of scepticism assisted possibly and perhaps unfairly by the recollection of another "discovery" by the same investigator, namely, the spirochæte of yellow fever, which was eventually proved to be the well known *Leptospira icterohæmorrhagiæ* of Weil's disease. What, then is the present state of knowledge concerning the ætiology of trachoma? Actually little or nothing definite, save the value of hygiene in prophylaxis, has been learnt during the past century. Surely a pathetic admission!

THE USE OF "NOVASUROL."

"NOVASUROL" or merbaphen is a drug which produces profuse and rapid diuresis when it is injected by either the intramuscular or the intravenous route. It is a double salt containing sodium oxymercuri-orthochlorphen-oxylacetate. Its mercury content is about 34%. There is no doubt whatever of its value in certain circumstances and reports have appeared from time to time in this journal of its value in œdema due to cardiac and renal disease. Its use, like that of all powerful drugs, is not altogether free from danger. Moorhead held in 1926 that the initial dose of one to two cubic centimetres, suggested by the manufacturers, should not be used, as it often caused diarrhœa and collapse. He thought that the initial dose should not be more than 0·5 cubic centimetre. Its use is contraindicated in certain acute conditions. It is of interest to note that D. H. Sprunt has published from the Department of Pathology of the Yale University a small series of observations which serve to emphasize the need for caution.[1] The drug was given to nine patients. Three were suffering from conditions contraindicating its use; one suffered from fever, one from tuberculosis and one from chronic nephritis. In two of these three instances necrosis of the renal epithelium was found at autopsy. The initial dose in each was one cubic centimetre. Further records of *post mortem* findings should be made. Sprunt's conclusion that "Novasurol" may be used as a last resort is a wise pronouncement. The initial dose must be small.

[1] *Archives of Internal Medicine*, September, 1930.

Abstracts from Current Medical Literature.

MEDICINE.

Parathyreoid Glands.

J. C. HOYLE (*The Practitioner*, May, 1930) discusses the relationship of the parathyreoid glands to disorders of bone. Collip showed that an active extract of the parathyreoid glands raised the blood calcium content by withdrawal of calcium into the blood stream from the soft tissues and bones. Decalcification of the bones occurs with excessive administration of the extract or excessive activity of the glands. Osteomalacia may be due to defective supply of calcium and vitamin *D* or seclusion from sunlight. Such patients may be cured by the administration of calcium in the diet along with irradiated ergosterol, cod liver oil or ultra-violet irradiation. Osteomalacia in adolescent girls and pregnant women may be cured by oophorectomy, though the rationale of this is not fully understood. In some instances osteomalacia is due to a disorder of the parathyreoid glands, such as hypertrophy or tumours. In these cases the blood calcium content is raised, whereas in other varieties of osteomalacia it is decreased. If a suitable diet, including irradiated ergosterol, does not rapidly result in relief to such patients, an exploratory operation for parathyreoid tumour is indicated. *Osteitis fibrosa* is closely allied to this type of osteomalacia. Tumour or hyperplasia of the parathyreoid glands often occurs and removal of such tumours may cure the disease. In these patients the quantity of serum calcium may be as high as twenty milligrammes *per centum* before operation and may fall to normal after operation. If most of the parathyreoid tissue is removed, tetany may occur; this may be relieved by giving a diet rich in calcium, with several grammes of calcium carbonate or calcium lactate daily, assisted, if necessary, by Collip's "Parathormone." Hyperparathyreoidism does not occur in *osteitis deformans*. Parathyreoid hypertrophy may occur in severe rickets, the result being an increased blood calcium, even at the expense of the already decalcified bones. Parathyreoid treatment may be helpful in delayed union of fractures, probably owing to its production of a condition of hypercalcæmia, the calcium being derived from the bones and deposited in the callus at the site of fracture. When lead is absorbed over long periods it is stored in the bones; it is liberated when large amounts of acid or alkalis are administered, the insoluble tri-lead phosphate, $Pb_3(PO_4)_2$, being converted into more soluble derivatives by slight changes towards the acid or alkaline side of the body reaction. Such treatment also mobilizes calcium from the bones, and it has been shown that active parathyreoid extract (Collip) in a dosage of 55 units daily over a period of three days increases the excretion of lead from an amount of one or two milligrammes to six or even ten milligrammes per day. At the same time calcium excretion is increased and blood calcium content raised. Owing to the increased quantity of lead and calcium in the blood, lead toxæmia, as evidenced by the existence of colic, palsy, encephalopathy, and hypercalcæmia with tetany must be watched for. Determinations of the quantity of calcium in the blood should be made if this method of treatment is used. A diet low in calcium should be given and acids, ammonium chloride or potassium iodide should be administered. If toxic symptoms occur, a diet rich in calcium with calcium medication will facilitate temporary storage of lead in the bones. Chronic hypoparathyreoidism may occur spontaneously, but is usually due to removal of parathyreoid tissue. This has three main features: Liability to acute tetany, especially during pregnancy or lactation; the frequent occurrence of cataract, and defective calcium deposit in the teeth during the period of growth. Parathyreoid hypertrophy normally occurs during pregnancy, the blood calcium is maintained at a high level and the mother's reserves of calcium become available to the infant. If the mother's blood calcium content falls below a certain level, tetany may occur. In chronic parathyreoid deficiency cataract is primarily due to some other cause, the calcium deposit being merely secondary.

Ayerza's Disease.

GEOFFREY L. S. KONSTAM (*The Lancet*, October 12, 1929) discusses Ayerza's disease in its various aspects. In reviewing the history of the disease he states that Abel Ayerza, of Buenos Ayres, drew attention in 1901 to the morbid entity of "cardiacos negros." The term "Ayerza's disease" was first used in 1909. Ayerza has left no written account of his observations, but if we are to accept his pupil Arrillaga's writings as embodying the teachings of his master, Ayerza's claim to a place in medical nosology must be based on his discovery of the "black cardiac" syndrome due to syphilis of the lung and pulmonary arteries. Both sexes are equally affected. The typical symptoms usually occur between twenty and fifty years of age. Cough and expectoration may have been present for many years. Gradually dyspnœa, cyanosis, persistent headache and drowsiness become added to the picture. Orthopnœa is the rule and hæmoptysis common. Congestive heart failure as a rule supervenes before death. The disease usually runs a protracted course. Unless emphysema is too great, systolic pulsation in the pulmonary artery can be seen and palpated and the pulmonary second sound is accentuated. The red blood cells number between five and ten million per cubic millimetre. The heart shadow, as observed radiologically, is much enlarged, both to the right and the left, giving a globular appearance, and the enlarged pulmonary artery is seen vigorously pulsating below the aortic knuckle. Shadows radiating from the lung roots may be due to vascular engorgement or fibrosis. An electrocardiogram will indicate a striking right ventricular predominance. In discussing the differential diagnosis the author stresses the fact that any disease process leading to pulmonary hypertension may produce symptoms clinically indistinguishable from Ayerza's disease. The differentiation from other forms of cyanosis, especially erythræmia (Vaquez-Osler disease), may be difficult, but reliance must be placed upon clinical and radiographic evidence and on the presence of a positive Wassermann reaction and the absence of enlargement of the spleen. Antisyphilitic remedies should be used in treatment. In the presence of persistent headache or frequently recurring hæmoptysis, venesection is useful. Oxygen may also be valuable. The author describes an illustrative case in which the pathological findings are discussed by Professor Hubert M. Turnbull.

The Bruit D'Airain.

A. J. GRANVILLE MCLAUGHLIN AND A. S. DIX-PERKIN (*The Lancet*, December 28, 1929) state that the *bruit d'airain* is not constantly obtained in pneumothorax. The case records of fifty patients in whom artificial pneumothorax had been induced, were analysed by the authors. Repeated examinations of the patients who developed effusions into the pneumothorax, showed that in general the *bruit d'airain* became more intense as the effusion became absorbed and as the pleural membrane became drier and more rigid. In a dry pneumothorax cavity with the lung completely collapsed and the pleura hardened, the *bruit d'airain* was the only metallic phenomenon to be heard, but if a collection of fluid was present in the pneumothorax cavity, the succussion splash as well as the *bruit d'airain* was elicited. Amphoric breath sounds were heard when the lung was partially collapsed and had an area of consolidation around one of the bronchi, when an uncollapsed pulmonary cavity was present and when a broncho-pleural fistula had formed. In no instance were amphoric breath sounds heard in the absence of the *bruit d'airain*. When in addition secretion was present in the lung, tinkling crepitations were heard.

Bilateral Avulsion of the Phrenic Nerves.

LASAR DUNNER (*Deutsche Medizinische Wochenschrift*, November 15, 1929) comments on the extensive literature which has been published since the value of the surgical treatment of certain types of pulmonary tuberculosis has been acknowledged. After mentioning that bilateral artificial pneumothorax has been a

recognized beneficial procedure for some years and that alternate pneumothorax and phrenic nerve avulsion have also been employed until recently, he states that he now performs bilateral phrenic nerve avulsion in selected cases where bilateral artificial pneumothorax cannot be induced. The fact that pneumothorax cannot be induced, indicates the presence of extensive pleural adhesions which will prevent the domes of the diaphragm from rising too quickly after the nerve avulsions. As a further precaution he advocates waiting for three weeks after the avulsion of the nerve on one side to observe the effects by means of X rays, especially the height to which the paralysed side of the diaphragm rises. Advanced and extensive bilateral disease is, of course, unsuitable for this method of treatment. A moderate rise of the diaphragm is not a contraindication, because in cases of bilateral pneumothorax it is astonishing to see with what little available lung tissue quiet respiration can be maintained. As a final precaution the activity of the other thoracic muscles of respiration must be ascertained prior to avulsion. The procedure is not as severe as it may seem and very little dyspnœa results, even in sick patients. However, it should be reserved for specially selected cases of bilateral disease, as a last resort.

Suprasellar Tumours.

T. DE MARTEL (*La Presse Médicale*, April 5, 1930) discusses the diagnosis and treatment of suprasellar tumours. These tumours arise near the *sella turcica* and spread upwards into the cranial cavity, pushing up the optic chiasma and giving rise to bitemporal hemianopsia and primary optic atrophy without deformation of the sella. Suprasellar meningioma arises near the tubercle of the sella, it occurs in middle age, giving rise to defective vision, more pronounced in one eye. The general health is not affected and there are no other symptoms nor signs, except bitemporal contraction of the visual fields, which is not observed unless specially looked for. A slight or marked pallor of one or both optic discs may be observed. A lateral skiagram reveals a normal sella, a stereoscopic skiagram reveals a larger and denser tubercle of the sella than normal. By these signs the diagnosis is made. Hypophyseal adenoma is the commonest cause of primary optic atrophy and bitemporal hemianopsia in middle-aged subjects, but in this condition obvious functional pituitary disorders occur, such as Babinski-Fröhlich syndrome and acromegaly; and enlargement of the sella is noted by X rays. Suprasellar adenomata occur that give rise to a clinical picture similar to that of the meningiomata, and the differential diagnosis is made only at operation. X ray examination, however, may reveal slight destruction of the posterior clinoid processes. Congenital cysts developing from the remnants of the cranio-pharyngeal canal are called Rathke's pockets; they cause symptoms in infancy or childhood. Calcium deposit occurs in the wall of the cyst and may be detected in a stereoscopic skiagram, though recognition may be difficult. The cyst may be as big as an orange or as small as a pea. Rarely calcification occurs in meningiomata and adenomata; this may confuse the diagnosis. The diagnosis of these tumours depends on careful and exact examination of the optic disc, the visual fields and the X ray examination of the *sella turcica*.

Lathyrism.

H. STOTT (*Indian Journal of Medical Research*, July, 1930) discusses the occurrence of lathyrism in the United Provinces and reports the result of a feeding experiment. Lathyrism is not a rare disease in certain areas of the United Provinces. It is most commonly seen in times of famine when lathyrus pea forms a greater proportion of the diet than in times of plenty. Animals are not immune, though fowls and pigeons are apparently never affected. The horse is especially susceptible. The main symptoms of equine lathyrism are weak lumbar muscles, "roaring" as a result of recurrent laryngeal palsy, dyspnœa, rapid, weak pulse, debility, tremor and stiffness of legs. Some crops of pea apparently cause lathyrism while others do not. Anderson, Howard and Simonsen found that while the pea *Lathyrus sativus* contained no alkaloid and was harmless, the pea *Vicia sativa* contained a poisonous alkaloid, divicine, which produced a characteristic and fatal disease when inoculated into guineapigs. Ducks fed on *Vicia sativa* died and monkeys exhibited nervous and muscular symptoms when this species of pea was added to their diet. Stott fed three ponies, one on a normal diet, one on a normal diet *plus* three pounds of *Vicia sativa* daily, and the third on normal diet *plus* three pounds of *Lathyrus sativus* daily. At the expiration of a period of four months and twenty-four days the animals were all in perfect health and had put on weight. Stott makes a plea for further research in this important problem.

Recoveries from Leprosy.

OSWALD E. DENNEY, RALPH HOPKINS AND FREDERICK A. JOHANSEN (*American Journal of Tropical Medicine*, March, 1930) discuss briefly the histories of sixty-five patients released from a leprosarium during the previous ten years. It is recognized that restoration of anatomical structure and physiological function after destruction by a leprous process is impossible, but within recent years many patients have been discharged from leprosaria following the arrest of the progress of the disease and disappearance of bacilli and most of the objective and subjective symptoms. Of the sixty-five patients discussed by the authors twelve had suffered from nodular leprosy on admission, their average age was 39·5 years and the average duration of their illness 3·7 years. The average period of hospital treatment for this group was 5·8 years. Twenty-six patients had mixed leprosy on admission; their average age was forty years, and they suffered for an average period of 6·8 years prior to segregation; the average duration of their hospital treatment was 5·5 years. The remaining 27 patients were affected with anæsthetic leprosy; their average age on admission was 30·9 years and they suffered from the disease for an average period of 6·5 years prior to segregation. The average duration of their stay in hospital was nine years. Crude chaulmoogra oil administered orally and intramuscularly was the drug in most constant use, but numerous other therapeutic measures were also used. The authors state that a critical evaluation of medicinal treatment is impossible owing to the many factors involved.

Blood Pressure.

S. C. MUELLER AND G. E. BROWN (*Annals of Clinical Medicine*, June, 1930) report observations on hourly rhythms in blood pressure in persons with normal and elevated blood pressures. Studies were made of 61 patients with essential hypertension and 26 with normal blood pressure. Systolic and diastolic pressures were taken hourly, day and night. Physical and mental activity was encouraged in the day time. Emotional effects were noted in nervous subjects when awakened that blood pressure observations might be taken, but averages were made of the results as a whole. The trend of the curves was similar in normal and abnormal subjects. The blood pressure was highest between 3 p.m. and 7 p.m. as a rule and lowest at 3 a.m. There was a slight rise as a rule after meals. The systolic and diastolic readings followed one another fairly closely in all variations.

Bronchial Asthma.

I. S. KAHN (*Annals of Clinical Medicine*, May, 1930) reports a case of severe bronchial asthma in which recovery followed the intravenous injection of 0·12 to 0·18 millilitre of a one in one thousand solution of adrenalin chloride repeated every thirty to forty-five minutes for four doses. In this instance death was imminent; repeated subcutaneous injections of adrenalin solution and morphine had been given without relief, and the patient was pulseless and unconscious. After each intravenous injection of adrenalin, improvement was noted, and eventually sleep resulted. Nausea, headache, pallor and vomiting occurred immediately after the injections, but these symptoms soon passed off. At a later date similar treatment was successful after seven intravenous injections at similar intervals. This treatment is recommended only in very severe asthma when there is no response to treatment by the usual methods.

Special Articles on Diagnosis.

(Contributed by Request.)

XX.

APPENDICITIS.

THE commonest incentive to surgical interference in the abdomen is a diagnosis of appendicitis. There usually follows the reflection that a life has been saved or at least a troublesome diseased organ removed. With feelings of regret, on the other hand, frequent and, may I say, unwelcome visits are received from unfortunate dyspeptics and neurasthenics who have been subjected to the popular operation of surgical removal of the appendix. The diagnosis in an ordinary case is usually easy, but in the exceptional ones may be exceedingly difficult. Errors in fœtal development, vagaries in site and a host of complications provide a wide variation of symptoms and signs.

Morphological Considerations.

The *processus vermiformis* is morphologically and structurally portion of the general caecal pouch, which at a certain stage of intrauterine life ceases to develop at the same rate as the caecum. *In utero* we find it accompanying the caecum normally from a position to the left of the mid-line in its rotation upwards and to the right, so that by the third month it lies up under the liver. Descending from this position, it usually reaches the right iliac fossa before birth. Owing to adhesions of a peritoneal developmental kind it may be arrested at any part in its progress. It may remain on the left side of the abdomen (Lockwood), even without transposition of the viscera. It may reach the subhepatic region and the ascending colon fail to develop. It may descend rapidly to its normal site in the right iliac region, even at the fourth month, if it gets a clear run, and perhaps drop with the caecum into the pelvis if the mesocolon is long.

Site with Normally Placed Caecum.

In the absence of adhesions the only fixed part of the appendix is at its attachment to the caecum underlying McBurney's point, 3·75 centimetres (one and a half inches) from the right superior iliac spine along the spino-umbilical line. Knowledge of its position may be obtained by X ray examination after a barium meal, for the latter mixes with or replaces the ordinary fæcal contents of the appendix in a large percentage of cases.

With the normally distended caecum the appendix, proximally at least, is hidden behind that organ, but with an empty, contracted blind gut it may be seen projecting from the lower part. The separation of the longitudinal muscular coat of the caecum does not involve the appendix with whose longitudinal coat the tæniæ are continuous. The appendix is never absent; shrunken remains may be found on careful search. Its length varies from five millimetres (Huntington) to 33 centimetres (Grauer) and is on the average 8·0 to 8·75 centimetres (three to three and a half inches). Traction on the elastic organ at operation may help to establish fallacious records.

The relative proportions of mucous membrane, lymphoid and muscular tissues and peritoneum vary in the adult. In the first couple of years of life there is very little lymphoid tissue. After this, until the age of twenty years the relative amount increases. The meso-appendix may extend over the whole length of the organ or more or less of the distal portion may be free and completely covered by peritoneum. Excessive fat in the mesenteriolum is often associated with the atrophic or *obliterans* type. Free from adhesions the appendix may be found coiled up or kinked behind the caecum or radiating in any direction from its fixed point, hence the description according to the position of the hands of the clock or the points of the compass.

Special Sites.

Owing to the difficulty in ascertaining relative figures, one finds it difficult to use the terms "usual" or "unusual" in regard to site. Retrocolic appendices are more prone to disease, hence they may be thought by some surgeons to be most common. The following special sites may be mentioned: (i) Retrocolic, whether free, in a pouch or fixed in the cellular tissue; (ii) mesenteric, either above or below the mesentery of the terminal ileum; (iii) pelvic, hanging over the brim or in the pelvis with a mobile caecum; (iv) subhepatic or high, due to incomplete descent of the caecum; (v) left-sided appendix, due to transposition of the viscera, to error in rotation or to a mobile colon.

Pathology.

Kinks, strictures and foreign bodies may play a mechanical part in the causation of appendicitis, but the infection is usually due to *Bacillus coli communis* or, more rarely, to a streptococcus, staphylococcus, the tubercle bacillus *et cetera*. Patients have ascribed it to a blow locally and to the pressure of the rope round the body on the tow path.

Date, plum and cherry stones may be simulated by concretions moulded in its lumen. Small shot, pins, threadworms *et cetera* have been found therein. A sandy-haired patient paid the penalty for chewing his moustache of being blessed with a perforation at the tip of the appendix through which bristly, short, sandy hairs protruded.

The clinical picture is greatly dependent on the intensity and extent of the inflammatory condition and on the position of the appendix. The inflammation may be acute or chronic. There may be relapses and recurrences. In fact, the latter are so frequent that "interval" operations are justified.

Acute Appendicitis.

Acute appendicitis appears in various grades of severity: (i) Catarrhal or interstitial inflammation without special peritoneal involvement; (ii) appendicitis with localized peritonitis with or without abscess; (iii) appendicitis with general peritonitis, sometimes following abscess.

Among the worst complications are thrombosis of the mesenteric veins and gangrenous cellulitis, occurring especially with the retrocolic appendix, perhaps leading to portal pyæmia. The typical case begins with pain in the epigastric region, gradually spreading downwards and ultimately settling in the right iliac region. Vomiting often accompanies this. Constipation is usual, but diarrhœa often occurs. Symptoms of toxæmia follow, including rise of temperature, rapid pulse and flushed face. Sweating occurs. Rigors indicate severe infection. At the first examination there are usually found: (i) Tenderness in the right iliac fossa, especially about McBurney's region; (ii) resistance of the right abdominal muscles on palpation; (iii) hyperæsthesia to light touch in this region, more extensive if the peritoneum is involved; (iv) Polymorphonuclear leucocytosis up to 15,000 or 20,000 leucocytes per cubic millimetre of blood. The abdomen becomes tumid.

As localized peritonitis develops, a definite, tender, fixed swelling may be felt, more marked if abscess formation takes place. The swelling may be movable when the tip of the appendix is wrapped in omentum. Fluctuation is rarely felt in the presence of an abscess, as the condition is usually diagnosed before this may be elicited. An exception may be when abscess has formed early in the pelvis and before it has burst into the bowel, bladder or peritoneal cavity.

With more diffuse peritoneal infection the pain becomes generalized over the lower half of the abdomen, respiration becomes more thoracic, tenderness and involuntary rigidity are more extensively felt. The abdomen is more distended, vomiting is frequent and the toxæmia is increased. Pulse rate and temperature are often fallacious guides. Despite gangrene or perforation the temperature may be subnormal and the pulse normal in rate, though weak. The use of morphine not only causes deception as to symptoms, but also as to pulse, tenderness and local resistance. Opiates should not be given unless operation is shortly to be performed, or if one is a convert to the non-operative cult.

Most deceptive is the rare variety in which with soft, flat abdomen, subnormal temperature and slow pulse, and without definite tenderness, the sunken-eyed, sick-looking patient presents at operation a gangrenous appendix with little peritoneal reaction. The result is usually fatal.

Differential Diagnosis of Acute Appendicitis.

In severe cases the problem is that of "the acute abdomen." Consideration in an article of this kind must of necessity be scrappy, as the differential diagnosis covers so many diseases. First, there are the many pathological conditions leading to local and general peritonitis, especially perforation or strangulation of certain viscera. The ultimate result in many instances is general peritonitis, the symptoms of which will not be reiterated. It is a far cry from a perforated gastric ulcer to a ruptured tubal gestation, but in difficult cases these may have to be considered.

Perforated gastric or duodenal ulcer is usually, but not always, preceded by typical gastric symptoms. There may be no such history in other instances. The pain is usually high in the abdomen, but may soon spread downwards. Severe initial collapse is evident. On percussion hepatic dulness may be absent. Under the X rays the sight of a subphrenic gas bubble affords confirmation.

Perforation of the intestine by foreign bodies, spreading ulcer (as in enteric fever, diverticulitis *et cetera*) causes peritonitic signs. The history and possibly the X rays may help to discover a foreign body. Appendicitis with its typical signs may develop during the course of enteric fever.

Internal strangulation of the intestine by bands, by Meckel's diverticulum, through gaps in omentum or mesentery, by kinks, volvulus *et cetera*, comes into the picture. The onset is sudden, the collapse often severe and the pain usually central and colicky. Pyrexia and leucocytosis are absent. There is obstruction to the passage of intestinal contents, so that distension may occur early.

Strangulated hernia in the right inguinal region is associated with the above symptoms of obstruction with central colicky pains. The local signs of hernia are present unless there has been reduction *en masse*. An inflamed appendix amongst the hernial contents may make diagnosis difficult.

Torsion of an undescended testis may escape consideration, if the scrotal content is overlooked. If the testis can be felt in the inguinal canal, mistake is unlikely, but if its position is intraabdominal, the pain is lower than the usual appendix pain. Local muscular resistance and thickening are absent. There is no pyrexia at first.

Diverticulitis resembles left-sided appendicitis in its symptoms and signs. If there is a history of the passage of blood and mucus with left iliac pain, we may suspect this disease. A skiagram taken after a barium enema, if the patient's condition permits, may show diverticula of the sigmoid.

Diseases of the gall bladder (cholecystitis, gall stones *et cetera*) and subhepatic appendicitis are often difficult to separate in diagnosis. In ordinary cases the right subcostal pain shooting through to the shoulder blade, with tenderness, resistance and perhaps a swelling under the liver, moving with respiration, will help. History of gall stone colic with perhaps jaundice may be available. If time permits, cholecystography may indicate gall bladder disease. However, the two diseases are often associated, so that if suspicion of this combination exists, ample incision should be made at operation for diagnosis and treatment.

Certain diseases of the kidney and ureter accompanied by pain require to be diagnosed from appendicitis. Calculus in the kidney or ureter may cause sudden pain in the loin or right iliac fossa accompanied by pyrexia and vomiting, if pyelonephritis is present. Pain shooting down to the external inguinal ring or even into the testis may be present in appendicitis. Frequency of and pain during micturition and perhaps blood-stained urine may be present when the inflamed appendix is in the pelvis. There is no leucocytosis unless with kidney infection. No local thickening can be felt. X ray examination will usually reveal the shadow of a calculus, but not always.

When there is a movable kidney with Dietl's crises, the kidney may be felt to be large and tender, if relaxation of the muscles is possible. The pain may, however, be simulated in retrocolic appendicitis. Pyelography will solve the problem, if not urgent. Perinephritic abscess may slowly develop without definite cause, or it may be due to kidney or appendix trouble.

Meckel's diverticulitis so closely simulates appendicitis that differential diagnosis may be impossible until operation. Usually pain is situated in these cases at a higher level.

Acute pancreatitis causes pain and tenderness more towards the subcostal region. The presence of cyanosis with fatty stools and a history of gall stone colic may help.

Tumours of the caecum may progress quietly until some ulceration or infection arises to cause confusion in diagnosis. The previous history of loss of weight, perhaps cachexia, the presence of a movable swelling and the absence of leucocytosis may be of assistance.

Tuberculous peritonitis in some of its many forms may cause symptoms resembling subacute appendicitis. There is usually a history of previous ill health, perhaps also the presence of foci elsewhere. Fluid perhaps may be discovered in the abdominal cavity.

In colitis the pains are more diffuse and, with tenderness, may follow the course of the colon. In many instances so-called "mucous" colitis has been cured by the removal of a diseased appendix. In the presence of nervous symptoms and in neurasthenics a diagnosis of appendicitis in this regard should be carefully "weighed in the balance," as it may be "found wanting."

Influenza with gastro-enteric symptoms may be complicated by appendicitis. Unless definite local signs of the latter are present, symptoms such as pain in the right iliac fossa or of more general character should be ascribed to the former. So in malaria there may be similar pains, but the history and blood examination will help in the decision. Abscesses in the abdominal wall or more deeply in the iliac fossa are usually not accompanied by such intestinal disturbance as is caused by appendicitis. Their onset is different, their progress more gradual and symptoms less serious.

Peritonitis due to streptococcal or pneumococcal infection may resemble the later stages of appendicitis. Without a definite history of the latter disease operation may alone enable the appendix as the causative factor to be eliminated.

In women ruptured tubal gestation, torsion of the pedicle of a right ovarian cyst and salpingitis may have to be considered. In instances of ectopic gestation there is usually, but not always, one or more missed menstrual periods, sudden pain in the lower abdomen, signs of shock and hæmorrhage, and metrorrhagia. There is no pyrexia or leucocytosis. Examination *per vaginam* may reveal general bogginess about the uterus. When an ovarian cyst with twisted pedicle is present, a defined swelling may be felt arising out of the pelvis, tender to the touch and distinct on bimanual examination. In the early stage diagnosis may be easy, but when peritonitis has ensued, the presence of a tender swelling to the right of the uterus with pyrexia may cause difficulty. Salpingitis is usually preceded by some cause such as abortion with infection, or gonorrhœa. Both tubes are affected, but a marked pyosalpinx to the right of the uterus makes diagnosis difficult.

Pleurisy, pneumonia and food poisoning are dealt with later.

To sum up, we must rely chiefly on the mode of onset, tenderness and resistance in the right iliac region with leucocytosis. At the same time the vagaries in position of the appendix must be borne in mind.

Chronic Appendicitis.

In chronic appendicitis there is as a rule the history of an acute attack of greater or less degree. Usually the association of some pain or discomfort in the epigastric region with slight pain in the McBurney region directs our attention to the appendix. One is sceptical as to the tactile skill of those diagnosticians who can always feel a diseased appendix and "roll it under the fingers." Margins of gut and muscle fibres are apt to deceive. Any doubts as to the causation of various troubles by chronic appendicitis are dispelled by the macroscopical and microscopical examination of the offending organ and by the relief of symptoms resulting from the ablation of the appendix. A striking example came under my notice. A young man called me in the early hours of the morning

owing to the sudden onset of pain in the appendiceal region. For some months he had been "a bit out of sorts" and complained that his tennis had been "rotten" owing to his feeling slack. He had had no pain until the morning of his operation. A sausage-shaped, non-adherent appendix containing about two drachms of pus was removed.

Appendix Dyspepsia.

Chronic appendicitis is undoubtedly a cause of reflex dyspepsia. It is exceedingly difficult in many instances to diagnose this class from gastric or duodenal ulcer and from the reflex dyspepsia of gall bladder disease. In typical examples the appendix may be considered to be the culprit, if the epigastric pain comes on immediately after or at irregular times after meals, if there is no lull in the symptoms as is usual in a patient suffering from ulcer, if the pain is not or is only partly relieved by alkalis, if nausea is present and vomiting does not afford some relief, and especially if, with these symptoms, palpation over the region of the appendix elicits tenderness and perhaps at the same time causes pain in the epigastrium. X ray examination helps to settle the difficulty. Bastedo's inflation of the colon with air is said to produce the right iliac pain and even the pain in the epigastrium, and so confirm the diagnosis.

The dyspepsia of chronic cholecystitis closely resembles that of chronic appendicitis, the feeling of distension being more marked in the former condition. The area of tenderness is in the gall bladder region. The X rays with a barium meal may show some deformity of the duodenal cap, but more certainly the absence of gall bladder shadow in cholecystography helps to settle the question, unless both diseases coexist.

Visceroptosis combined with the presence of peritoneal membranes and bands produces symptoms, such as pain in the right iliac region and intestinal stasis, suggestive of chronic appendicitis. When a neurasthenic condition is also present, one must be careful to avoid a rash decision in diagnosis. Many of these appendixless patients haunt the surgeon or his *confrères* for years. X ray examination after barium meal and enema may reveal the real site of obstruction and elicit tenderness there. In one instance, after the removal of a diseased appendix enveloped in adhesions for recurrent attacks of right iliac pain, the pain returned and was not relieved till a stout band of "Jackson's membrane" type kinking the ascending colon was divided.

Spondylitis in the lower dorsal or lumbar region must be considered in those patients with chronic pains referred to the right lower part of the abdomen and with slight tenderness. An X ray photograph may save the patient from an unnecessary operation.

Radiological Examination.

In these chronic cases X ray investigation is most valuable for diagnostic purposes. With the barium meal increase in size and frequency of the waves of peristalsis with a rapid emptying of the stomach may suggest reflex irritation from the appendix or other abdominal organ. The position of the appendix filled with barium may be seen. Stasis of the barium in the appendix suggests disease. Tenderness on pressure directly over the visualized organ is in my opinion positive evidence of disease. Again, an appendix that does not admit barium suggests a diseased condition. Deformity of the filled organ is also informative. Distortion of the adjacent hollow viscera may be seen, due to kinking or bands of adhesion, the result of previous disease.

Appendicitis in Children.

For two years after birth the lymphoid tissue of the appendix is scanty. Also the omentum does not attain its full development in childhood. Hence appendicitis which is fortunately rare in infancy, is much more dangerous owing to weakness of the local defence, general peritonitis being common. Early diagnosis is therefore most important. With older children we may get some assistance from the history of the illness. Crying interferes with proper abdominal examination, so that we are often compelled to induce light anæsthesia with chloroform. This is very helpful and may permit of the discovery of local muscular resistance or thickening. At the same time examination *per rectum* should be made, when the finger may discover bogginess or thickening high up. The hand on the abdomen with the finger in the rectum may bimanually detect a thickening, swelling or even fluctuation when the appendix is in the pelvis. In older children rectal examination may be made without anæsthesia. Sometimes pressure over the left iliac fossa may cause pain in the McBurney region, probably owing to the disturbance of gas pressure within the bowel.

The position adopted by the little patient may help us to decide the site of the inflamed appendix. Both thighs may be held flexed to relieve the abdominal pain. If the right hip only is flexed and the child cries when the thigh is straightened, there may be involvement of the psoas muscle by extension from a retrocolic appendicitis. Crying on rotation at the right hip joint may suggest spread to the adjacent *obturator internus* from the appendix in the pelvis. The signs and symptoms are much the same in children as in adults, but as a rule the child is sicker, the pulse faster and the temperature higher.

On the whole diagnosis is most difficult when the appendix is in the pelvis, but the child may help if the pain can be located in the suprapubic or lower abdominal region, if dysuria and frequency of micturition are present, or if there is pain in the rectal region with the passage of blood-stained mucus with or without fæces. In one instance, that of a girl of twelve years, I was deceived for twenty-four hours by the fact that her first menstrual period coincided with an attack of gangrenous pelvic appendicitis. The increasing toxæmia with rising pulse and temperature led to the discovery of the real cause at operation. The mesenteric type is dangerous, leading to early and rapid progress of toxæmia with frequent vomiting and quickly distending abdomen. On the other hand, the disease in the pelvis has a greater tendency to become localized.

Differential Diagnosis of Appendicitis in Children.

Differential diagnosis presents special features in children owing to the greater prevalence of certain diseases in childhood. In gastro-intestinal disturbance from undigested food or irritants, or in gastro-enteritis, vomiting usually comes on early before pain and the pain is usually central and colicky. Between the spasms of pain the abdomen may be soft to palpation. Diarrhœa often accompanies the spasms. Leucocytosis of any pronounced degree is usually absent.

In acute pyelitis, which practically always occurs in girls, the onset is heralded by fever or rigor and toxæmic symptoms followed by pain. The pain is usually not very acute at the onset, occurs higher than the ordinary appendiceal site and may be in the loin. Tenderness is also high, towards the region of the kidney pelvis. Muscular resistance is not great. Even in the absence of pain and frequency of micturition the urine is cloudy from bacilluria or pyuria. Microscopical examination settles the diagnosis if blood and pus cells are found in the urine. An exception to this finding may occur in the later stages with a blocked ureter.

In regard to cyclic vomiting, more and more of the class of patient with a condition previously diagnosed as cyclic vomiting will be found to be suffering from appendicitis. The sickly child with recurrent attacks of epigastric pain or discomfort, with acetone odour of breath and acetonuria should be examined at the earliest onset of an attack for tenderness in the right iliac region. Complete cessation of the attacks and restoration to good health may be hoped for when such tenderness is found and the appendix removed.

In mesenteric lymphadenitis in the ileo-caecal region infection may occur from appendicitis. In other instances the cause is unknown. Frequently the glands are tuberculous. There is right iliac pain recurring from time to time, with tenderness and resistance about the McBurney region. Sometimes the rounded glands may be felt. In these instances of recurrent subacute lymphadenitis diagnosis may perhaps only be determined at operation.

In intussusception there is no difficulty in diagnosis when the signs are well developed. There is the onset

of sudden abdominal pain with vomiting accompanied by shock with associated pallor *et cetera*. The attacks of pain are of intense colicky character with intermissions. The passage with tenesmus of blood-stained mucus of red currant jelly appearance is characteristic. Finally, the sausage-shaped tumour, ultimately perhaps felt *per rectum*, is distinctive, though a general anæsthetic may be required for proper abdominal and rectal examination. In the rarer forms in which no gross tumour is discovered, and especially when inflammation has occurred, the difficulty of diagnosis is increased.

In right-sided pleurisy and pneumonia the presence of right-sided abdominal pain and tenderness obscures the diagnosis, especially with regard to inflammation of the high appendix. Physical signs in the chest may be absent when the trouble begins deeply. The diaphragm may be involved from below in appendicitis. Thus in doubtful cases above and below the diaphragm we may have rapid and distressing respiration with working of the *alæ nasi*. Perhaps pressure on the left side of the abdomen causing pain on the right side may incline us towards appendicitis. Until definite thoracic physical signs have developed diagnosis may remain doubtful without operation.

JOHN RAMSAY, C.B.E., M.S., F.C.S.A.,
Honorary Consulting Surgeon, Public Hospital, Launceston.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE SOUTH AUSTRALIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Darling Building, University of Adelaide, on September 25, 1930, DR. C. E. C. WILSON, the President, in the chair.

Tuberculosis.

DR. E. BRITTEN JONES showed a patient suffering from tuberculosis of the adult type. The child, aged eight years, had come under observation in April, 1929, with a history of cough of six months' duration, profuse expectoration, loss of appetite, loss of weight and general fatigue.

On examination the right lung was found to be extensively involved. There was evidence of cavitation in the uppermost lobe. No abnormal physical signs were detected in the left lung. Tubercle bacilli were found in the sputum. X ray examination revealed extensive involvement of the right lung. There were some calcified areas in the region of the hilus of the left lung, but no shadows suggestive of active disease.

Induction of artificial pneumothorax was undertaken in May, 1929. Adhesions in the upper part of the chest prevented complete collapse of the uppermost lobe. However, for some months the partial collapse caused great improvement, although cough was still present. A small quantity of fluid appeared after some months of treatment and shortly afterwards it was found impossible to maintain the collapse already attained. Apparently a pleurogenous fibrosis had been set up and the introduction of air under very high pressure (up to +36) had no appreciable effect on the thickened lung tissue. It was then considered advisable to perform phrenic avulsion, which procedure was followed by only a temporary improvement in the cough and decrease in expectoration. Consequently Dr. L. O. Betts performed thoracoplasty, the second stage being completed on July 11, 1930. The patient stood the operation remarkably well. As a result of the latest procedure there was great clinical improvement. At the time of the meeting there was no cough, no expectoration, no feeling of fatigue, no pyrexia, and the patient was heavier.

Thoracoplasty is a drastic procedure in a child, but the prognosis, being so grave, justified the operation, which was followed by such improvement.

Injury to the Foot.

SIR HENRY NEWLAND showed the radiograms of a woman who had suffered an injury to the foot sustained by falling down stairs. She had been told that a sesamoid bone of the big toe had been fractured. An examination of the radiogram, however, showed that there was one normal sized sesamoid bone and a small fleck beside it which he considered was the representative of the normal second sesamoid. This view was confirmed by clinical examination, as the tenderness was over the dorsal aspect of the metacarpo-phalangeal joint, not in the sole over the sesamoid bone.

Carcinoma of the Transverse Colon.

DR. C. T. CH. DE CRESPIGNY showed a specimen of carcinoma of the transverse colon which was adherent to the gall bladder. It had formed a fistula into the duodenum. The symptoms complained of during life were vomiting and copious diarrhœa causing dehydration and wasting.

Leprosy.

DR. C. DUGUID showed a female patient who was suffering from leprosy. This report will be published in a subsequent issue.

Tuberculosis in Children.

DR. R. L. THOROLD GRANT read a paper entitled: "Tuberculosis of the Lungs and Bronchial Glands in Children" (see page 679).

DR. E. BRITTEN JONES congratulated Dr. Grant on his able and illuminating lecture. He considered the particular advantage of a positive reaction to the Mantoux test was that it drew attention to tuberculosis in the household. The type of adult tuberculosis commonly met with (phthisis) represented a reinfection of patients who had had a primary complex in early life. "The tuberculous child of today is the phthisical adult of tomorrow," hence the importance of guarding such children from the possibility of frequent and massive infections. He considered a positive reaction to the Mantoux test with obvious calcified lesions was of favourable import provided care was taken in safeguarding the patient from frequent and massive reinfection. One fact that must be borne in mind was that occasionally there was no reaction to the intradermal test, even in chronic tuberculosis.

As regards physical signs, he had found of value a sign described by French writers as being the result of a "fissuritis." This consisted of submammary crepitant sounds, the result of an interlobar pleurisy with subsequent adhesions. Calcified abdominal glands were not infrequent. He had seen such evidence of tuberculosis in seven instances during the past three years. The majority of the patients had a scar in the appendix region.

He further mentioned the case of a child of a tuberculous mother. The infant had been under observation from birth and the Mantoux test was carried out at three months and at six months with no result; however, at nine months the reaction was positive and the child had at this stage enlarged cervical glands which proved ultimately to be tuberculous. The mother was warned of the danger of infection, but would not heed the advice given, that the only safe course to take was to send the child to friends or relatives.

The overwhelming majority of early childhood infections was the result of close and intimate contact, and it was the duty of the physician to trace the infection to its source.

DR. C. DUGUID, in thanking Dr. Grant, said he felt it was unfortunate that so much money was being spent in cancer research and so little in the problem of tuberculosis. Cancer was a disease of late life, but tuberculosis attacked the child and the youth. Both from the humanitarian and the economic aspect it was to be hoped governments some day would spend more on the eradication of tuberculosis. Twenty-five years ago pulmonary tuberculosis headed the list of causes of death in Scotland, whereas today it was well down in the lower half. City and county tuberculosis medical officers with their teams had done

thorough work, but there was another factor. Sufferers from pulmonary tuberculosis in Scotland rarely lived beyond the age of thirty years. In Australia it was not uncommon to find grandparents affected. The handling of this human source of infection was going to prove a root problem in Australia.

DR. J. W. BROWNE complimented Dr. Grant on his thoughtful and judicious paper. He thought that Dr. Grant had covered most of the ground adequately, but in the matter of prophylaxis perhaps might have devoted more time to the *Bacille Calmette Guérin* vaccine. This method was still *sub judice* and it would probably be advisable to content oneself in the meantime with watching results elsewhere. Dr. Grant was right in stressing the importance of keeping in touch with contacts. In Dr. Browne's opinion many of the tuberculous infections met with in later life were due to the reactivation of old foci which had originated in childhood.

Many studies in sensitization by means of the Mantoux or von Pirquet test showed that the entry of even one infected child into a school previously free from infection, or of even one infected immigrant into an uninfected village often meant that quite a noticeable proportion of the contacts developed a positive reaction, and this, too, even if the newcomer did not suffer from open or active disease. This observation showed how complicated the problem of dealing with contacts might become. It also threw some light on the problem of explaining the very slight fall in the incidence of pulmonary tuberculosis in Australia during the past decade despite a considerable fall in the death rate. Each State should have a properly constituted tuberculosis bureau. Boards of health could do something and general practitioners more in dealing with this difficult problem, but their efforts should be coordinated by a bureau.

MEETING OF THE FEDERAL COMMITTEE.

A MEETING OF THE FEDERAL COMMITTEE OF THE BRITISH MEDICAL ASSOCIATION IN AUSTRALIA was held on October 2, 1930, at the British Medical Association House, 135, Macquarie Street, Sydney, SIR HENRY NEWLAND, C.B.E., D.S.O., in the chair.

Representatives.

The following representatives of the Branches were present:

New South Wales: Dr. J. A. Dick, C.M.G., Dr. R. H. Todd.
Queensland Branch: Dr. D. G. Croll, C.B.E., Dr. A. Murphy.
South Australian Branch: Sir Henry Newland, C.B.E., D.S.O., Dr. Bronte Smeaton.
Tasmanian Branch: Dr. Gregory Sprott.
Victorian Branch: Dr. F. L. Davies, Dr. J. Newman Morris.

Dr. Gregory Sprott and Dr. R. H. Todd acted as proxies for Dr. A. W. Shugg, of the Tasmanian Branch, and Dr. D. D. Paton, of the Western Australian Branch, respectively.

Minutes.

The minutes of the previous meeting of March 28, 1930, copies of which had been sent to the members after the meeting, were submitted and signed as correct.

Financial Statement.

The financial statement for the six months ended June 30, 1930, prepared by Messrs. Coates, Cunningham and Company, Chartered Accountants, and audited by the Honorary Auditor, Dr. W. H. Crago, together with the Australasian Medical Congress (British Medical Association) accumulated funds account, was presented and adopted.

The Medical Officers' Relief Fund.

The report of the trustees of the Medical Officers' Relief Fund (Federal) was read and received.

Australasian Medical Congress (British Medical Association).

The financial statement of the Third Session of the Australasian Medical Congress (British Medical Association) was received.

A letter was received from the Western Australian Branch nominating Dr. D. D. Paton as President of the Fourth Session of the Congress. The nomination was approved and Dr. Paton was appointed.

An advance of £100 was made to the Executive Committee of the Fourth Session of Congress.

Model Rules for Ethical Procedure.

At the time of the meeting of the Federal Committee in March, 1930, the draft model rules governing procedure in ethical matters for Branches in Australia had been approved by the several Branches after the further revision undertaken on the suggestion of the Central Ethical Committee, and they had again been forwarded to the Central Ethical Committee, where they received attention. They had been returned by the Central Ethical Committee after being approved by that body in their revised form. The communication of the Central Ethical Committee, signifying its approval, was read and received, and it was resolved:

> That the Branches be advised of the approval of the Central Ethical Committee of the Council, with a view to the model rules being adopted by the several Branches.

Post-Graduate Study in England.

The Honorary Secretary read a reply from the Medical Secretary of the Association to a letter which the Committee at its last meeting had directed him to send, explaining that it was thought that further extension of post-graduate teaching in England would be appreciated by visiting members from Australia and that the members taking advantage of the opportunities for study would increase in number.

The reply, which was dated July 28, 1930, was in the same terms as a letter of the same date addressed by the Medical Secretary to the Council of the Victorian Branch, which was published in THE MEDICAL JOURNAL OF AUSTRALIA of September 27, 1930, at page 453.

Conference of Oversea Representatives.

At the previous meeting of the Committee consideration was given to a proposal of Dr. E. R. Fothergill, a member of the Council of the Association, that a conference of representatives of Federal Councils and oversea Branches and Divisions should be held in England every five years. The Medical Secretary had been communicated with and asked for advice in regard to the proposal and his reply was read. He pointed out that the matter had not had the consideration of the Council, but it was hoped that something of a special nature would be done in this way at the centenary of the Association which was to be celebrated in 1932. It was decided that in the circumstances no further action should be taken.

Federal Council.

Further consideration was given to the proposals for the formation of a Federal Council for the Branches in Australia. The draft Memorandum and Articles of Association and By-Laws were published in THE MEDICAL JOURNAL OF AUSTRALIA of January 18, 1930, at page 92. At the previous meeting of the Committee consideration was given to recommendations and requests for alterations received from the Victorian and New South Wales Branches, and it was arranged that copies of the Constitution as amended should be sent to the several Branches for the formal approval of each and that on the receipt of the formal approval of all of them it should be submitted to the Council of the British Medical Association in accordance with the requirements of the British Medical Association By-Laws, By-Law 23 (2).

The Honorary Secretary reported that replies approving of the amendments had been received from all the Branches

except the New South Wales Branch. The letter from the New South Wales Branch had to do with Article 14. This article read:

> The number of members of the Executive Committee inclusive of *ex officio* members shall not be less than three nor more than six.

The New South Wales Branch again suggested, as it had done before, that in Article 14, (i) the word "not" be omitted, also the words "less than three nor more than"; and (ii) words be added to the effect that "no more than one member representing any one Branch shall be a member of the Executive Committee," so as to make the article read: "The number of members of the Executive Committee, inclusive of *ex officio* members, shall be six; and no more than one member representing any one Branch shall be a member of the Executive Committee." During the discussion it was pointed out by the Honorary Secretary that provision existed for the appointment of a substitute if a member of the Executive Committee of the Council was unable to attend.

Dr. D. G. Croll raised the question of the expense of sending members to attend meetings several times in the year.

Dr. J. Newman Morris was opposed to the interstate view taken by the New South Wales Branch; he felt that no Branch should object to the Executive Committee having a preponderance of members from any one State.

It was resolved, in accordance with the first proposal of the New South Wales Branch that the word "not" be omitted, also the words "less than three nor more than," so that the article read: "The number of members of the Executive Committee, inclusive of *ex officio* members, shall be six." The further proposal of the New South Wales Branch, namely, that words be added to the effect that no more than one member representing any one Branch shall be a member of the Executive Committee, was not approved. It was resolved:

> That subject to the alteration of Article 14, the number of members of the Executive Committee, inclusive of *ex officio* members, shall be six, and that, being approved by the New South Wales Branch, the Constitution be referred, on behalf of the several Branches concerned, to the Council in accordance with By-Law 23.

Annual Subscription to the British Medical Association.

At the last meeting of the Federal Committee it was resolved that the Central Council of the Association be approached with a view to the reduction of the amount of the subscription payable on behalf of members in Australia from £1 5s. 6d. (£1 11s. 6d. less 6s. capitation allowance) to £1 1s. This had been done and the matter was to be considered by the Organization Committee at its next meeting. The Medical Secretary had in the meantime written asking that the Committee might be supplied with the reasons that had led the Federal Committee to ask for the reduction. The Honorary Secretary's reply, stating the position for the information of the Organization Committee, was read and approved.

Scholarships and Grants (1930).

A communication from the Medical Secretary was read, in which he advised that a research scholarship for one year as from October 1, 1930, had been granted by the Council to E. S. J. King, M.S., B.S., 1923, M.D., 1926 (Univ. Melbourne), F.R.C.S. (England), 1927, Dr. King's subject being "Endometrioma of the Ovary."

Repatriation Department.

In 1924 an arrangement was made by medium of the Federal Committee and the several Branch Councils for friendly society lodge medical officers to attend widows and orphans of soldiers whose death was due to war service, and widowed mothers of such deceased unmarried soldiers. The payment of mileage and other such incidental expenses by the Repatriation Department in cases where the beneficiary of the scheme was unable to do so, had been urged upon the Department by the Federal Committee and at the previous meeting a subcommittee had been appointed to approach the Department. This had been done and a letter, dated July 11, 1930, from the Chairman of the Repatriation Commission, was received advising that the Commission was unable to approve of the payment of incidental expenses as suggested by the Federal Committee, and expressing the view that it was a matter entirely for private arrangement between the friendly society lodge medical officer and the beneficiary concerned, as in the case of friendly society lodge members.

Australian Soldiers' Repatriation Act, 1920-29.

At the last meeting of the Federal Committee consideration was given to new legislation concerning two new appeal tribunals and the effect of this legislation on information given to the Commission upon a confidential basis, and it was resolved that the matter should be discussed with the Commission and that the Repatriation Department should be asked to inform medical practitioners that, when clinical notes were asked for, all such information was available by the ex-soldier whose case was being considered.

A reply had been received from the Department to the effect that the matter had been considered, that it was assumed that no information would be disclosed without the permission of the medical practitioner and that the Department had no knowledge of instances in which the practice of the Department had acted detrimentally. It was decided that the matter should be taken up with the Department again and that the request be pressed that when a medical practitioner is asked for clinical notes about a repatriation applicant, the practitioner should be informed by the Department that the notes will be available for the applicant to see.

Dispensing of Medicines by Repatriation Department Local Medical Officers.

A letter from the Chairman of the Repatriation Commission, dated September 18, 1930, was received, dealing with the question of dispensing medicines by local medical officers. He drew attention to the existing "Instructions for Local Medical Officers," paragraph 21, which provides that "where there is not a pharmacist in business and the local medical officer does his own dispensing, he may dispense for ex-soldiers and claim the same rates of payment therefor as are allowed the pharmacists," and asked that approval be given by the Federal Committee to a proposal for Repatriation local medical officers to "be instructed that in places where there is a pharmacist in business within two miles, or less, from their surgery, the pharmacist is to undertake the dispensing of all prescriptions." He explained that this was in accordance with an arrangement with the pharmaceutical societies throughout the Commonwealth and the concurrence of the British Medical Association was sought. The Committee decided that the Repatriation Commission be advised that the Committee had no objection to the proposed alterations being embodied in the "Instructions to Local Medical Officers."

Medical Services to Natives in Central Australia.

At the last meeting of the Federal Committee further consideration was given to the question of disease among the aborigines in Central Australia, and Dr. J. Newman Morris and Dr. F. L. Davies were appointed a subcommittee to investigate the question and report to the next meeting. The report of the subcommittee was read.

Appreciation of the report and of the work of the subcommittee was expressed, and the Honorary Secretary laid before the Federal Committee certain information that he had that day received from the Chief Medical Officer of Northern Australia. After discussion it was decided that further action be deferred until the next meeting and that in the meantime each member of the Committee be furnished with a copy of the report.

Health Research Council.

Further consideration was given to the matter of the establishment of a health research council, as recommended by the Royal Commission on Health in 1925. A letter on the matter had been sent to the Federal Minister

for Health in May, 1930, and a reply received to the effect that the time was not considered opportune for the establishment of such a Council. The matter was allowed to stand over until the next meeting.

Remuneration of Ships' Surgeons (Overseas Ships).

The question of the remuneration of ships' surgeons in ships trading between Australia and overseas ports was again mentioned and discussed. Attention was drawn to the fact that the knowledge required by ships' surgeons was becoming more technical than it had been and that, on some lines especially, qualified medical men were engaged for the work. The shipping companies in Australia, however, who engaged medical practitioners for ships not under the Commonwealth *Navigation Act*, did not require special services from them, and in a large number of cases the ship's surgeon gave his services in return for the trip and without remuneration. In the circumstances there was little hope of obtaining better remuneration for those who devoted themselves to that form of medical practice. The Overseas Shipping Representatives' Association was not concerned with the engagement of ships' surgeons and there did not appear to be any association of overseas shipping interests that was. The matter was allowed to stand in abeyance.

The Australian Inland Mission Frontier Medical Service.

The Queensland Branch had forwarded a scheme that had been submitted to it by the Australian Inland Mission in regard to a frontier medical service. The scheme was forwarded as a basis of discussion. Dr. Newman Morris put before the meeting a later scheme of the Australian Inland Mission. After discussion it was resolved:

> The Federal Committee recognizes the necessity for more adequate medical service in sparsely settled districts of the far north of Australia. Medical service is not effective without aerial transport and improved wireless and telegraphic facilities. The Committee approves of the purpose of the Australian Inland Mission scheme and is prepared to assist in forming a suitable scheme.

Duty on "Insulin."

The Victorian Branch forwarded a letter from Burroughs Wellcome and Company, Limited, protesting against the imposition of 30% duty on "Insulin" of British production. The Committee was of opinion that in the interests of sick people the recently imposed duty on "Insulin" should be removed. It was decided to communicate with the Department with a view to the removal of the duty.

Date and Place of the Next Meeting.

It was determined that it be left to the Chairman to fix the date and place of the next meeting.

Thanks.

Votes of thanks were accorded to the Chairman, Sir Henry Newland, for presiding at the meeting, and to the New South Wales Branch Council for the accommodation provided for the meeting.

Jubilee of the New South Wales Branch.

The Chairman drew attention to the fact that the New South Wales Branch was celebrating the fiftieth anniversary of its foundation, and it was resolved:

> That the congratulations and good wishes of the Federal Committee be conveyed to the New South Wales Branch on the celebration of its jubilee and the opening of the new house, Macquarie Street, Sydney.

NOMINATIONS AND ELECTIONS.

THE undermentioned have been nominated for election as members of the New South Wales Branch of the British Medical Association:

Drew, William Robert McFarlane, M.B., B.S., 1930 (Univ. Sydney), Sydney Hospital, Sydney.

Black, John Roland, M.B., Ch.M., 1926 (Univ. Sydney), Wellington.

MacKellar, Duncan Gordon, M.B., B.S., 1930 (Univ. Sydney), Port Kembla.

The undermentioned have been elected members of the New South Wales Branch of the British Medical Association:

Bohrsmann, Gustav Temple Hall, M.B., B.S., 1927 (Univ. Sydney), Enmore Road, Enmore.

Bolger, Thomas Natal, M.B., Ch.M., 1926 (Univ. Sydney), Lewisham Hospital, Lewisham.

Costello, Kathleen, M.R.C.S., 1929 (England), L.R.C.P., 1929 (London), 27, Hazelbrook Road, Wollstonecraft.

Heaslip, William Gordon, M.B., B.S., 1929 (Univ. Adelaide), Salamo, *vid* Samarai, Papua.

Lyon, Marjorie, M.B., B.S., 1928 (Univ. Sydney), 104, Louisa Road, Long Nose Point, Balmain.

Taylor, Clive William, M.B., B.S., 1929 (Univ. Sydney), 8, Alpha Street, Willoughby.

Medical Societies.

THE MEDICAL SCIENCES CLUB OF SOUTH AUSTRALIA.

A MEETING OF THE MEDICAL SCIENCES CLUB OF SOUTH AUSTRALIA was held at the University of Adelaide on July 4, 1930.

Vegetable Protoplasm and Environment.

DR. R. H. PULLEINE spoke on the reaction of vegetable protoplasm to environment. He pointed out that the macrophyllous vegetation of the rain forests was succeeded by sclerophyllous xerophytes in the drier areas, each succession being more xerophytic, leaves giving way to phyllodia and phyllodinous to aphyllous forms, such as *Acacia continua*. The ground vegetation accompanying these in Australia was, he explained, largely halophytic.

Dr. Pulleine pointed out that the succulent manifestation of xerophytic plants is poorly marked in Australia, only a few almost cosmopolitan *Ficoidæ*, *Crassulaceæ* and *Portulacaceæ* showing it. On the other hand, such a tendency is abundantly illustrated in the northern and southern isothermic regions in America and Africa. In its marked form it postulates the suppression of the vegetative organs into a succulent stem of water storing capacity. This in some families, especially *Ficoidæ*, is associated by the mucilaginous character of the protoplasm, its actual and potential water content rising as high as 99% in some species of *Mesembryanthemum* and perhaps 95% to 97% in some opuntias. Both in Africa and America there is development of root succulents, stem succulents, leaf succulents, and in these countries the convergence of two unrelated families, *Euphorbiaceæ* and *Cactaceæ* is most marked. Thus the landscapes of southern Africa and northern Mexico show succulent euphorbias and *Cerei* respectively, which imitate each other with great fidelity.

Specimens of plants showing convergence, high water content *et cetera* were used by Dr. Pulleine to illustrate the address.

DR. DAVIDSON said that many of the plants exhibited by Dr. Pulleine appeared to be so highly specialized that they were unable to adapt themselves to marked changes in their environment. He asked whether there were some forms of the groups as a whole which were more adaptable to marked environmental change and whether they gave rise to a wider distribution and greater variation in form.

Dr. Pulleine stated that such forms did occur.

Dr. Davidson asked what the temperatures inside the lens portion of the *Fenestraria* sp. would be under conditions of normal surface sun temperature in its normal habitat.

MR. WOOD said that the temperature would probably be a few degrees higher than that of the surrounding atmosphere.

Mr. Wood pointed out that the carbohydrate metabolism follows a peculiar course in succulent plants. In particular, pentosans are stored and not starches. These are usually mucilages and capable of absorbing considerable quantities of water. Also in respiration complete oxidation of carbohydrates does not occur, but usually stops at an intermediate stage and organic acids accumulate. He also pointed out the homologous forms taken by *Euphorbia* and various *Cactaceæ* under similar environmental conditions and contrasted Lamarckian and genetical viewpoints.

Read's Formula for Estimating the Basal Metabolic Rate.

DR. GILBERT BROWN described some of the methods used in examining patients in order to grade them as to their suitability for the administration of general anæsthesia. In addition to the physical examination there were three simple calculations which had proved trustworthy—Moot's rule, the energy index and the breath holding test.

Moot's Rule.

The pressure ratio, a fraction having the pulse pressure as numerator and the diastolic pressure as denominator, may be normal between 40% and 60%. If the ratio is either high or low, there is reason to apprehend danger. If the ratio lies between 25% and 75%, the condition is probably operable; if outside these limits, it is probably inoperable. Miller investigated this in a series of 1,000 patients and found a death rate of 3·23% among those with operable and 23·07% among those with inoperable conditions.

The Energy Index.

The sum of systolic pressure and the diastolic pressure multiplied by the pulse rate is the energy index. The normal lies between 13,000 and 20,000; any readings outside these limits are considered as indicating bad risks.

The Breath Holding Test.

In the breath holding test the patient sits perfectly quiet for five minutes, then draws a full but not abnormally deep, inspiration. The breath is then held, with the mouth closed and the nostrils compressed with the fingers, while the time is noted. The normal time for which the breath can be held in this manner is thirty to forty seconds. The patients are graded as follows: (*a*) When the breath is held for under ten seconds, the patient is unfit for any general anæsthetic. (*b*) When the breath is held for ten to eighteen seconds, the patient may be fit for short gas anæsthetics. (*c*) When the breath is held for eighteen to thirty seconds, the patient presents a moderate risk. (*d*) When the breath is held for over thirty seconds, the patient is probably suitable for anæsthesia.

Lantern slides were shown illustrating the application of these tests.

In some patients, however, it is desirable to estimate also the basal metabolic rate. Several attempts have been made to produce a formula by which the basal metabolic rate can be calculated from the pulse rate and blood pressure. The formula evolved by Read and published in 1922 has been tried both in England and America and has been further simplified until it now reads:

$$\text{B.M.R.} = \tfrac{3}{4}(\text{P.R.} + \tfrac{3}{4}\text{P.P.}) - 72$$

where P.R. is the pulse rate and P.P. is the pulse pressure.

Read himself found that there was only an error of 10% in 60% of cases and of 20% in 91% of cases. Hunt found that there was only an error of 10% in 54% of his cases. The figures that Dr. Gilbert Brown had been able to obtain in Adelaide were very few, but had been within 10% of error. The opinion of the members was asked as to whether sufficient reliance might be placed on the formula to be of real service.

Lantern slides were shown of the blood pressure charts made during operations for exophthalmic goître and the results reviewed by comparing them with the estimation made with Read's formula. In one patient operated upon under ether anæsthesia in 1923, there appeared little depression of the blood pressure, but death took place within thirty-six hours. The formula gave the basal metabolic rate equal to + 84%. In another patient the blood pressures had been greatly depressed during the operation and death took place two and a half hours later. The formula in this patient showed the basal metabolic rate equal to + 100%. This latter case emphasized three points: First, the operation was unwise in this patient when so ill and without better preparation; second, that the operation performed was too extensive and ought to have been stopped when the blood pressure fell so greatly; third, that ether anæsthesia was inadmissible in very severe toxic goîtres and that nitrous oxide and oxygen or ethylene and oxygen should be used. Several lantern slides were exhibited in which it was shown that the blood pressures were very little depressed by operation for exophthalmic goître when the gas anæsthetics were employed.

The College of Surgeons of Australasia.

ELECTION OF FELLOWS.

A MEETING of the Council of the College of Surgeons of Australasia was held at Sydney on September 29, 1930. Fifty-two applications for fellowship were presented to the Council. Thirty-two of these applicants were admitted to fellowship. The names are as follow.

New South Wales: C. G. H. Blakemore, P. D. Braddon, J. A. F. Flynn, D. S. Foy, G. R. Halloran, I. D. Miller, H. P. Pickerill, H. A. Ridler, M. P. Susman, A. B. K. Watkins.

New Zealand: P. A. Ardagh, W. J. Barclay, L. A. Bennett, H. S. Billcliff, H. K. Christie, R. A. H. Fulton, A. McGregor Grant, W. C. McCaw, J. A. Pottinger, J. L. Reed, G. D. Robb.

Queensland: M. Geaney, A. C. F. Halford, H. S. McLelland, M. G. Sutton.

United States of America: R. J. Hardstaff.

Victoria: D. Bird, H. S. Bush, L. Doyle, W. E. A. Hughes-Jones, J. Love, L. J. Middelton.

The following applicants were admitted as junior members:

New South Wales: H. M. de Burgh, R. G. S. Harris, K. S. Parker, Brooke Moore, T. Y. Nelson, M. H. Thomas, C. J. M. Walters.

Victoria: W. R. D. Griffiths.

Hospitals.

THE ROYAL ALEXANDRA HOSPITAL FOR CHILDREN.

THE following report on some issues of blood compatibility tests has been received from Dr. Frank Tidswell, Director of Pathology, Royal Alexandra Hospital for Children, Sydney.

The discovery by Landsteiner in 1900 that the serum of some individuals would agglutinate the red blood corpuscles of others displayed the need for precaution in performing transfusion. There followed the comparison of various specimens of blood and their arrangement into types and blood groupings according to the well known classifications of Jansky and of Moss. At an early stage we became distrustful of these types, and have for long preferred to test the patient's blood directly against that of the prospective donor in each individual case when transfusion was contemplated. Amongst our now rather numerous records there are many tests of sufficient interest

to merit report because the patients and donors were consanguineous. It could have been supposed that blood of near relatives would have been compatible nearly always, but this has not proved to be the case.

In 251 tests in which the donors were father, mother, brother or sister of the patient, the specimens of blood were compatible in 161 and incompatible in 90 instances. In 94 tests against the father's blood 67 specimens were compatible and 27 incompatible; among 57 with mother's blood 34 were compatible and 23 incompatible; and among 20 with the blood of brother or sister nine were compatible and 11 incompatible.

Of 35 comparisons with the blood of both parents the results were as follow:

Patient's blood compatible with blood of both parents, thirteen instances.
Patient's blood compatible with blood of father only, nine instances.
Patient's blood compatible with blood of mother only, six instances.
Patient's blood compatible with blood of neither parent, seven instances.

It may be mentioned that on a few occasions the compatibility relationships have changed after transfusion, previously compatible blood becoming incompatible, as if the injected blood had acted like an antigen.

Quite clearly consanguinity affords no guidance to the likelihood or otherwise of blood compatibility, and it is not quite safe to use a previous donor without retesting. These issues emphasize the desirability of continuing our present practice of individual tests.

Obituary.

NEVILLE REGINALD HOWSE.

A FEW men achieve preeminence in the profession of their choice, still fewer carry their preeminence into a second sphere; those privileged to render distinguished service in a third field of activity are but seldom found. Such men are born to be leaders; they inspire confidence and they earn for themselves the appreciation of those of their own and of later times. Such was Sir Neville Reginald Howse, whose death was recorded in the issue of September 27, 1930.

Neville Reginald Howse was born at Strogursey, Somerset, England, on October 26, 1863. His father, Alfred Howse, was a medical practitioner and a member of the Royal College of Surgeons of England. He went to Fulland's School, near Taunton, and became a medical student at the London Hospital in 1880. In due course he graduated and became a member of the Royal College of Surgeons of England and licentiate of the Royal College of Physicians of London. After graduation he was appointed Demonstrator in Anatomy at the University of Durham.

He came to Australia on account of his health in 1889 and practised first of all at Newcastle, New South Wales, and then at Taree. In 1895, he visited England for post-graduate study. At the end of two years he gained his fellowship of the Royal College of Surgeons of England. He returned to Australia in 1898 and in the following year he went to Orange where he succeeded to the practice of the late Dr. Van Someren. It is well known that Howse thought seriously of taking up surgical practice in Sydney. It is equally well known that he met with much opposition from other members of the medical profession. There have been many highly qualified and capable surgeons in country districts in Australia and it cannot be said that Howse set a precedent by succeeding in becoming a surgeon of the first order practising in the country. It will be remembered, however, that the name "Howse, of Orange," came to be synonymous with sound surgery. Many another large country centre owes its surgeons and its recognition as a surgical centre to the example set by Howse.

Howse had not been long in practice in Orange when the South African War broke out. He was an imperialist, he had to be in the thick of it. Somehow he always gave the impression of being a typical Englishman, even after years of residence and practice among Australians. He went to the Cape with the New South Wales Lancers as Surgeon-Lieutenant. It was here that he won the most highly prized of all British honours, the Victoria Cross. The manner of his winning the decoration is well known. It is described in the military records of the State in the following short statement:

> During the action of Vredefort, 24th July, 1900, Captain Howse went out under a heavy cross fire and picked up a wounded man and carried him to a place of shelter. (Gazette, 4th June, 1901.)

He was soon promoted to be Captain and returned to Australia with the Queen's Medal and four clasps and the King's Medal and two clasps.

After his return from the South African War, Howse took up his practice in Orange with the vigour and concentration that were so characteristic of him. Reference has already been made to the reputation gained by him as a safe and reliable surgeon, but more than this, he became peculiarly part of the district, necessary to both medical practitioners and to the whole community, collectively and individually. The medical practitioners in that part of the State regarded him as their leader; they respected his surgical judgement, admired his technical skill, held him in the highest esteem for his personal qualities. The country as a whole recognized his worth and accepted his service. He was Mayor of Orange on more than one occasion. The individuals with whom he came into contact trusted him as all patients trust their faithful medical attendants. Howse had that in him, however, which proclaimed him as a confidant for the fallen, a support for the weak, an inspiration to the weary. He gave his undivided attention to the person or the problem of the moment. Small wonder was it that those amongst whom he worked had faith in him and loved him.

With the outbreak of war in August, 1914, it was but to be expected that one so fired with enthusiasm and zeal for his country and so naturally fitted to be a leader would be immediately to the fore. On August 10, 1914, the Commandant of the Second Military District communicated with Howse by telephone in regard to active service out of Australia. Howse, who at that time held the rank of Honorary Major in the Australian Army Medical Corps Reserve, reported on the following day to the Principal Medical Officer and accepted command of the medical unit then being raised to accompany the force for the Pacific Islands. Howse received the rank of Lieutenant-Colonel and the title of Principal Medical Officer of the detachment. He had as his second in command Captain F. A. Maguire. Howse thus took part in the first military operation undertaken by Australia during the Great War. Although it was thought that by being sent to New Guinea he was unfortunate, in that he might be sidetracked for a considerable period, those who held this idea did not know the character of the man. When the occupation of the German Territory in New Guinea was complete, Howse naturally looked for further directions in which to expend his energy. He obtained permission from the Administrator to return to Australia in order that he might enlist in the Australian Imperial Force. Permission was the more readily given in that Howse had as his second in command one who was qualified to carry on the existing organization in the Territory. On October 4 Howse left New Guinea in the *Berrima* and was able to report that up till the time of his sailing there had not been a single trace of serious illness among the two thousand men transported suddenly into a tropical country.

The Australian Imperial Force was mobilized rapidly and was to have sailed for Europe at the end of September, 1914. Owing, however, to the presence of German warships in the Pacific, the date of sailing was postponed. This was fortunate for Howse, and he was appointed, on the recommendation of Surgeon-General Williams, as "Supernumerary Medical Officer" attached to Headquarters. He was detailed to assist the Director of Medical Services

during the voyage and acted as his staff officer. On his arrival in Egypt, Howse found plenty to occupy his attention. His peculiar position made it easy for him to offer counsel when matters of moment were being discussed. His previous experience in the South African War doubtless had a great deal to do with the fact that the General Staff paid heed to him; his forceful personality, his sincerity, his insight and his knowledge of men could not fail to attract attention. It was impossible for him to remain long in a subordinate position. General Bridges recognized his worth and appointed him "Embarkation Medical Officer" when the troops were disembarking at Alexandria. Later on, when, at the time of the formation of the Anzac Corps, Colonel Ryan, Assistant Director of Medical Services of the First Australian Division, was transferred to Corps Headquarters, Howse was appointed to the vacant post. From this time onward Howse worked hard to improve the health of the troops. He did his best to see that hospital facilities were provided for the increasing number affected by disease of various kinds. He got things done. For example, he was convinced that certain dental work should be carried out for the troops. He was instrumental in having a request sent to Australia for the appointment to one of the Australian general hospitals of a dentist who was already doing good work in another unit. The request was refused. Shortly afterwards arrangements were made with the Assistant Director of Medical Services of the New Zealand Expeditionary Force that a New Zealand dental officer should be attached to the Australian unit.

In the preparations for the landing at Gallipoli Howse was a tower of strength. He acted on matters medical for the whole Anzac Corps. He landed with Divisional Headquarters on "the beach." He established a casualty clearing station and worked with the utmost devotion to duty. Dr. Archie Aspinall in his tribute has made reference to this part of Howse's military career.

The subsequent advancement of Howse to the elevated position he afterwards held is a long story. It is intimately bound up with the whole history of the Australian Imperial Force; and by the efficient service that he later on created he was instrumental in making the best use for the common cause of all the resources of the medical profession in Australia. There is no doubt that this will be shown clearly in the "Official History of the Australian Army Medical Services" that is shortly to be published. The need for a "D.M.S., A.I.F." was clearly seen in these early Gallipoli days. Various recommendations were made, the usual counter suggestions and recommendations resulted, and it appeared that nothing definite would be done until Colonel R. H. Fetherston, Director-General of Medical Services, Australia, visited Anzac. Colonel Fetherston saw the need, recognized that Howse was the only man for the job and the appointment of Howse as "D.M.S., A.I.F." was actually made by him. The appointment was confirmed in due course. It is recorded officially as having been made on December 1, 1915.

The efficiency of the Australian Army Medical Corps in the Great War, especially in the latter days, was the direct result of the work of Neville Howse. He built up the service and made it what it was. He knew it in detail; he knew what his officers could do, for he was a good judge of men. He had the happy knack of choosing as his assistants men whom he could trust, men who knew how to do their jobs and did them. This, of course, is a necessary quality in all men holding important administrative posts. His immediate assistants were inspired to do their work primarily by their leader's obvious devotion to the cause, but also by their personal regard, often amounting to affection, for him. He was approachable. He was just in his decisions. When he had to be severe he used no half measures. He might appear hard at times, for the exigencies of the service often demanded disregard of the individual for the sake of the efficiency of the machine. He was at heart, however, very human. A lieutenant-colonel in charge of one of his field ambulances had on one occasion to detail a certain officer for a dangerous duty. This officer was a medical graduate of high attainments, a man of considerable promise for the future in the world of science, a man held in high esteem by all his fellows. While carrying out his orders the officer was killed. The lieutenant-colonel was naturally very cut up about the loss of so fine an officer. He thought to himself that if the orders had been given in some other way, the loss of the officer might not have occurred; he allowed this to prey on his mind, and not knowing why he did it and not stopping to realize what he was doing, he sat down and poured out his heart in a private letter to Howse. The reply came promptly and was at once understanding and sympathetic. Here was no official red tape. And the story is but typical of the man.

There is no need to dilate further on the organization of the Australian Army Medical Corps under the guidance of Howse. Australian medicine, and particularly the surgical side of Australian medicine, owes a great deal to him for for his vision during the war period. Australia is in his debt for all time because of the efficient care of her wounded and sick sons during the dark days.

When Neville Howse returned to Australia, he came laden with honours. His Majesty the King had conferred the honour of Knight Commander of the Most Honourable Order of the Bath and Knight Commander of the Most Distinguished Order of Saint Michael and Saint George. He went back to his practice in Orange. After another visit to England he entered upon what may be described as the third phase of his life—his political career. He sought and obtained a seat in the House of Representatives as member for Calare. He achieved the unique distinction of gaining ministerial honours in his first parliament. As Minister for Defence he succeeded the Honourable E. K. Bowden. He proved himself an able administrator. In 1928, owing to ill health, he resigned the portfolio and took over the Department of Home and Territories and that of Health and Repatriation. He worked zealously in both spheres. Divergence of opinion over political matters is, of course, wide, and there are many who did not agree with either Howse's views or his political acts. There are none, however, who can call in question his honesty or sincerity of purpose. His loss of the seat for Calare at the time of the fall of the Bruce-Page Ministry is fresh in the memory of everyone.

After this Howse went to England. He was not well and he consulted Lord Moynihan who operated on him for gall stones. For a while it appeared that he had made a good recovery. When Lord Moynihan was in Canada attending the annual meeting of the British Medical Association, symptoms of the old malady made their appearance and Howse had to face surgical operation again, this time at the hands of Sir Hugh Rigby. The result was not satisfactory and he died on September 17, 1930. Much more might be written of him—of his love of sport, of the friendships that he made and of his home life—but it must suffice that we have attempted to portray him in his three principle rôles, those of surgeon, soldier and statesman. In all three he earned richly the gratitude of his fellow countrymen. This, we venture to hope, will mitigate in some small degree the grief of his widow, his three daughters, his two sons and his brother, Dr. C. B. Howse.

Dr. F. A. Maguire writes:

Neville Reginald Howse was intensely loyal to both the country of his birth and that of his adoption; he was one of the first to answer the call when the Empire was at war.

The South African War was the scene of his first experience of active service. From 1900 till 1902 he was an officer of the Australian Army Medical Corps, holding a commission as lieutenant and captain. During that time he had the high distinction of winning the only Victoria Cross ever granted to a member of an Australian medical service. In July, 1900, Captain N. R. Howse "went out under heavy cross fire, picked up a wounded man and carried him to a place of shelter." This action was an example of the coolness, determination and utter indifference to personal danger which was again and again displayed by Howse during the whole of his military service.

When the Great War of 1914-1918 broke out, Howse was in practice in Orange, New South Wales. He held the

honorary rank of Major, Australian Army Medical Corps Reserve, but he had not taken any active part in military affairs since 1902. On August 10, 1914, the Commandant, Second Military District, telephoned to Major Howse offering him an appointment with a force then being mobilized for active service abroad. Howse came to Sydney at once, where he interviewed the Principal Medical Officer, the late Colonel Fiaschi. The writer became associated with him for the first time on that day. Howse was offered the command of the medical detachment that was to accompany the Australian Naval and Military Expeditionary Force which, under the command of the late General William Holmes, was setting out to occupy the German possessions to the north of Australia. The one question that exercised Howse was, "Is it the big thing?" He was assured by the Principal Medical Officer that there was no immediate opportunity of active service elsewhere. This at once influenced his decision, for he had a consuming desire to be engaged in active military service.

His powers of organization had full scope here. In the week available he took all steps to insure that the equipment for the medical service of the Force should be complete; the troops were closely examined medically and dentally, and courses of instruction were put in hand for all ranks to prepare them for service in the tropics. Lieutenant-Colonel Howse, as Principal Medical Officer, spared no endeavour to safeguard the health of the troops, both on the voyage and after the occupation of German New Guinea. So well did he lay his plans and so efficiently were they administered that a force of "nearly two thousand troops were transported by sea and disposed on shore in a tropical country and in the first two months, up to October 15, 1914, not one single case of serious illness had occurred." This testimony was made by General Holmes when he gave permission to Lieutenant-Colonel Howse to return to Australia to seek further active service abroad. For Howse realized that "the big thing" was to be sought elsewhere.

On his return to Australia the Australian Imperial Force had been formed. The First Division had embarked and was on the point of sailing. All appointments had been filled. However, at the last moment Lieutenant-Colonel Howse was attached to Headquarters, First Division, as "Supernumerary Medical Officer" and detailed to assist Surgeon-General Williams for the voyage, acting as his staff officer. On the arrival of the convoy in Egypt the Australian Imperial Force, which was proceeding to England, was ordered to land in Egypt to train there. The Director of Medical Services, Surgeon-General Williams, however, proceeded to England and Lieutenant-Colonel Howse was appointed by General Bridges as Embarkation Medical Officer with a small staff to make the medical arrangements for disembarkation. By this time Lieutenant-Colonel Howse had established that cordial and intimate contact with the General Staff that is so essential for the smooth working of the administration of the medical services. "Possessed of great tact, insight, charm of manner, incisive and convincing address, and an ambitious resolve to make his presence felt," he laid the foundations of a career as an able administrator, a guardian of the interests of the medical services and the safeguard of the health of the troops that led him to the highest position in the medical services of the Australian Imperial Force.

On December 28, 1914, he was promoted to the rank of Colonel and appointed Assistant Director of Medical Services of the First Division. When, in April, 1915, the Australian Imperial Force went to Gallipoli, Colonel Howse foresaw the tremendous difficulties that would face the medical services. He made most strenuous efforts to make suitable provision for the casualties which he foresaw. That there were at times almost hopeless confusion and disorganization was in no way his fault. On the contrary, authorities argue that it was his presence and influence which evolved order out of chaos. The appreciation by Sir Brudenell White and the account of the official historian of the Australian Army Medical Corps make this abundantly clear.

Colonel Howse served through the campaign at Gallipoli. On September 11, 1915, he was appointed Deputy Director of Medical Services of the Australian and New Zealand Army Corps. He was at all times thinking ahead and foresaw the necessity for a unified control of the Australian medical services. But he was most active in encouraging his officers to do their utmost in safeguarding the health of the troops. The Anzac Medical Society was the outcome of his desire to stimulate the professional interests of the medical officers.

After the evacuation of Gallipoli the Australian Imperial Force was reorganized. Ultimately General Birdwood was appointed General Officer Commanding the Australian Imperial Force. As an outcome, a Director of Medical Services for the whole of the Australian Imperial Force in Egypt, France and England became a necessity. As the man who knew most about the problems, who had foreseen the necessity for the reorganization and who was best qualified from a military, professional and personal standpoint to hold such a position, Colonel Howse was selected. He was promoted Surgeon-General on November 22, 1915, and appointed as "Director of Medical Services, Australian Imperial Force." From then on to the termination of the war General Howse administered the medical services of the Australian Imperial Force. The high standard of efficiency that he set and maintained were recognized by His Majesty the King when in 1917 General Howse was made a Knight Commander of the Most Honourable Order of the Bath and in 1919 Knight Commander of the Most Distinguished Order of St. Michael and St. George.

As a soldier General Howse was the embodiment of the spirit of service. He never spared himself any exertion to carry out his duty. His first thoughts were always for the health of the troops. That they should be housed as well as circumstances would permit and cared for in sanitation, food and clothing at the highest possible standard was always his foremost thought. To this end he insisted on efficiency in those under his command. He had the gift of selection of men. Youth was in his eyes no bar to selection and promotion. He built up round him, as time and opportunity permitted, a staff of loyal, keen officers who were imbued with his ideas and through whom his influence was extended to every unit of the great force.

As a man, General Howse was always kindly, approachable, full of sympathy and encouragement for others and at all times willing and anxious to help any officer or man who appealed to him for assistance or support. He was always anxious to stimulate his officers to render themselves efficient both from a military and professional standpoint. Throughout Australia there are hundreds of medical men who bear most kindly memories of one whose personality radiated geniality, who was ready to recognize merit, who was most kindly and approachable by anyone who had need of advice, whose standard of duty and personal service was of the highest, and who never asked anyone to do anything he was not prepared to do himself or to go anywhere where he himself was not prepared to go.

Dr. Archie Aspinall writes:

If twelve men were asked to write about the late Sir Neville Howse the resulting records would be widely divergent, as he was a man of many parts. All would agree as to his tireless energy, ability and determination to excel in whatever he put his hand to. As a youth most of my holidays were spent on stations in the west of New South Wales and there "Howse of Orange" was a name to conjure with. Everyone knew of his surgical skill, humanity and dominating personality. Without knowing him personally, I had formed a very definite mental picture of him as a doctor who was a soldier and had won the Victoria Cross. On meeting him many years later the picture was not spoilt, but enhanced, for was he not tall, handsome and every inch a soldier? Living very close to and working with him at Anzac, I realized why he was loved by those who knew him best. Possessed of ready wit and a wide knowledge of the world, combined kindliness, good humour and an intense interest in the aspirations of younger men, talks with him were at once an education and an inspiration. The merry twinkle in his eye completely disarmed one, even though he spoke words of

censure. Once Sir Neville made up his mind, which did not take long, that a certain course of action was the correct one, he was indifferent to hostile criticism. His judgement of the characters of men was almost uncanny. He had to make many administrative changes. Some men were hurt thereby. Although he had lived in Australia so long, he was unmistakably an Englishman. He taught us that we were Australians first and foremost working for the common good—collectively important, but individually of little importance. This was a hard lesson for medical men fresh from civil practice to learn and caused some heart burning at times. Sir Neville was really very ill at Anzac, but few knew of it, as he was never off duty. I can see him now, walking up the hill from the beach to his "dug-out," pausing several times to get his breath, but refusing even to discuss his health, let alone accept any advice, saying the job had to be done. How well he did his job is only known to those who saw the chaotic condition of the medical service in Egypt before he had control.

Dr. A. E. Colvin writes:

Just what can I write of Neville Howse? Tongues far more gifted than mine have spoken and pens more inspired have written the deeds and the character of this man, one of the greatest and the bravest among men. But I must pay my tribute to one who was everything to me. I can only say that twenty years ago I commenced my life as a young medical man and met Neville Howse, and ever since he has been my guiding star, pointing out the path of duty in my profession, in war and in the civic works of peace. Any little thing that I have ever done that was worth while and was of any use to my country and my fellow men was due entirely to him and to the standard and example of service he set.

He loved the Empire and this country and his life was one of tireless, devoted service for both. He was a master of men, because he was master of himself and never admitted to any self weakness, but at the same time had the greatest sympathy and understanding for the frailties of others, who always felt safe and happy and comforted when he was near by and in control.

Today's English mail brings me messages from him, a day or two before he passed hence. He knew he was going and happily and bravely as ever faced the unknown and the great beyond. He spoke words of love and help and encouragement to us to "carry on" and do our duty in these days when so many of us are inclined to give up hope.

WALTER HENRY RUSSELL.

WE regret to announce the death of Dr. Walter Henry Russell, which occurred on October 30, 1930, at Semaphore, South Australia.

VALENTINE MACDONALD.

WE regret to announce the death of Dr. Valentine Macdonald, which occurred on November 5, 1930, at South Yarra, Victoria.

Medical Prizes.

THE ALVARENGA PRIZE.

THE College of Physicians of Philadelphia announces that the next award of the Alvarenga Prize, being the income for one year of the bequest of the late Señor Alvarenga, and amounting to about three hundred dollars, will be made on July 14, 1931, provided that an essay deemed by the Committee of Award to be worthy of the prize shall have been offered.

An essay intended for competition may be upon any subject in medicine, but must be accompanied by a written assurance from the author that it has not appeared previously in print, either in whole or in part in any form, and has not been presented elsewhere in competition for a prize. The essay should represent an addition to the knowledge and understanding of the subject based either upon original or literary research. It must be typewritten and in English acceptable for publication without necessity for editing by the Committee. Any illustrations should be appropriate and correctly annotated with the text. Essays must be received by the Secretary of the College on or before May 1, 1931.

Each essay must be sent without signature, but must be plainly marked with a motto and be accompanied by a sealed envelope having on its outside the motto of the paper and within the name and address of the author.

It is a condition of competition that the successful essay or a copy of it shall remain in possession of the College; and that it may be published by the author with the consent of the College; other essays will be returned upon application within three months after the award.

The Alvarenga Prize for 1930 has been awarded to Dr. Henry A. Harris, London, England, for his essay entitled: "Cod Liver Oil and the Vitamins in Relation to Bone Growth and Rickets."

Correspondence.

PUERPERAL SEPSIS.

SIR: As a medical student in the early eighteen-eighties I attended sixty-three maternity cases in the slums of Lambeth. In those days there was absolutely no antenatal supervision. The expectant mother applied at the hospital and received a card at any period of her pregnancy. She was not examined. When her labour began, the card was brought to the hospital and the resident obstetric clerk responded to the summons. He carried with him, thrown into a brief bag, a Higginson syringe to use for an enema or for douching the vagina, a silver female catheter, a perineal needle and silver and silk sutures, a bottle of liquid extract of ergot, and, an innovation in my time, a pot of carbolized vaseline. After my first case or two, being fastidious, I carried in addition a towel and piece of soap.

Of my sixty-three cases five were born before arrival. There was one capable nurse-midwife in the district who prided herself on the number of "born before arrival" cases which she succeeded in arranging for the students. "Born before arrival" cards counted just the same.

I had one terrifying case of *post partum* hæmorrhage which was, however, quickly controlled by hand grasp of the uterus, aided possibly by the administration of an extra two drachms of the liquid extract of ergot. Every case received two drachms on delivery of the placenta. I had summoned the resident *accoucheur* to this case, but the hæmorrhage had ceased when he arrived. I summoned him also to another case, one of protracted labour, in which I had "got the wind up." He came, examined, laughed at me and left. The midwife who assisted me did not believe that he was the resident *accoucheur* because he was not wearing the conventional top hat and black tail coat. I summoned him again to a third case, that of a woman who had had two still-births and was anxious to have a live baby. The resident *accoucheur* brought with him the assistant obstetric physician, the late Dr. Cory, who wished to test the advantages of Tarnier's axis-traction forceps, which were then new. The patient was anæsthetized, my catheter was smeared with carbolized vaseline and I was told to draw off the urine. This had to be done by touch, under the bedclothes. The forceps were then taken from Dr. Cory's bag, the convex surfaces smeared with carbolized vaseline and handed to me to apply, much to my gratification, and we were all pleased when the baby announced its arrival into the world, under the bedclothes, with the usual yell.

My remaining cases were ordinarily normal. Two cases are worth recording to indicate what I have meant by

the slums of Lambeth. In the first I was shown into an upstairs room and the door shut upon me. The patient was lying on a small quantity of straw in a corner of the room. The room was absolutely bare of furniture except for a chamber utensil. I had no assistance. It transpired that the midwife was in the house all the time, drunk, and put in an appearance after I had left. The patient, a *primipara*, was delivered, the chamber with placenta handed out to a neighbour tenant of the house, who emptied it and returned it to me filled with warm water. I washed the patient down, handed the chamber to the obliging neighbour and received it again to use for washing the baby and later, once more, for washing my hands. I washed and dressed the baby and left the house, having gained a new and useful kind of experience. When I visited the patient on the tenth day she came downstairs to let me into the house.

On another occasion I visited a patient on the tenth day, a Sunday, and found her sitting down to dinner with her husband and three older children, the baby being in her arms. The Sunday dinner consisted of a small crust or two of bread and a saucer of uninviting boiled rice for each of the five persons.

At another case, to pass the tedious time of waiting between pains, I occupied myself by killing with the blunt end of a lead pencil the bugs that inhabited the walls of the bedroom.

One point requires emphasis. All manipulations were carried out under the bedclothes. One was not supposed to view any portion of the patient's body. Hence slight perineal tears, if they occurred, were not recognized. I was fortunate enough not to experience a ruptured perineum. I use the word fortunate, because in my later years the prevention of a torn perineum has been the source of my greatest anxiety.

The procedure at a confinement was as follows: Arrived in the patient's room, one washed one's hands in a domestic manner; then, waiting with the forefinger of the left hand well greased with carbolized vaseline, one waited for the signs of an oncoming pain. When these came, the hand would be thrust under the bedclothes and would fumble for the vaginal orifice. The best way to make sure of this was by feeling for the anus and then running the finger along the perineum until it slipped into the vagina. On one occasion I had the mortification of being told by the lady patient that I was "in the wrong hole!" The anus was patent, the head emerging at the moment. A vaginal examination was made with every pain. If the finger encountered fæces, the assisting midwife was asked to wipe this away, while one turned one's back lest one should get a glimpse of the patient's "privates." And so on, hour after hour, until the head came through. (I had one breech case.) Then Credé's expression of the placenta, two drachms of ergot, an examination and washing of the baby's eyes, and "Good-bye, I will call to see you tomorrow." On the morrow questions as to the five "B's": bowels, bladder, breasts, binder and baby. I had only one temperature, 101°, on the occasion when I thought it necessary to douche the vagina; and I remember that the midwife thought and told me that I was unnecessarily officious. She appeared to think that I carried out this procedure in order to secure a look—it was indeed not easy to avoid seeing some portion of the lady's body, as the returning water had to be received into a chamber utensil.

Sixty-three cases, no deaths, no still-born babies, one forceps case, one *post partum* hæmorrhage, one with an offensive discharge for two or three days, and all under exceptionally unhygienic conditions, attended by a youth who had previously never seen a midwifery case, his only preparation being the assiduous attention to the lectures of the late Dr. Gervis, the best lecturer to whom I ever listened, and the reading and rereading of Leishman's text book, a preparation that was considered adequate in those days.

One's procedure is somewhat different now. One is allowed to see what one is doing. Briefly, I wear a gown; I do not wear gloves; I do not allow my patients to be shaved; I wash my hands surgically; I wash the pubes, vulva, perineum and anal region of the patient with carbolic soap and wipe off the superfluous lather; I palpate the abdomen; I wash my hands surgically again and make a vaginal examination. Then I wait, and perhaps make a further vaginal examination, possibly to push back an œdematous anterior lip. I administer chloroform in practically every case, and to *primiparæ* I have previously given a hypodermic injection, sometimes two, of Burroughs Wellcome and Company's "Hyoscine A." I have been lucky. I have had only one case of definite puerperal fever, a condition which I attributed, probably unjustly, to a "Gamp" midwife who assisted me. The patient recovered after an anxious fortnight that took a year off my life and for which I received three guineas.

A friend of mine, the most thorough practitioner I have known, was asked to attend a lady of good circumstances, who on the occasion of her two previous confinements had suffered from puerperal fever. A week before her expected date, ten days before labour actually began, the doctor installed his most trustworthy nurse in the house. The patient was shaved and made as surgically clean as possible. Iodoform swabs were inserted within the vulva, iodoform diapers were applied. These were changed after each operation of the bowels and after micturition, for ten days. Every known precaution was taken at the time of the confinement. No vaginal examination was made. The patient had an easy confinement and, after the usual interval of anxiety, puerperal fever developed.

Why did my sixty-three patients escape and not she? I don't know. Who does know?

Yours, etc.,

A. B. BROCKWAY,
M.R.C.S. (Eng.), L.R.C.P. (Lond.).
Redcliffe, Queensland.

PUBLIC MEDICAL OFFICERS OF NEW SOUTH WALES.

SIR: I have read with surprise your statements as to salaries of Public Medical Officers in New South Wales contained in an article on the Public Services in the Education Number of October 25. Your figures are in many cases erroneous and where correct are liable to convey a wrong impression to your readers.

The salaries of permanent whole-time medical officers employed by the New South Wales Government are governed as from July 1, 1927, by an agreement concluded in 1928 between the New South Wales Public Service Board and the Public Medical Officers' Association of New South Wales. This agreement has recently been continued subject to such variations as may be caused by variations in the State basic wage until July 1 next and provides for a minimum annual salary of £600 for males and £500 for females. The following extracts will give your readers some idea of its scope.

Senior Medical Officer of Health: £1,250 to £1,300 *per annum*.
Assistant Medical Officer of Health: £825 to £925.
Medical Officer of Health, Newcastle District: £900 to £1,000.
Director of Tuberculosis: £1,000 to £1,050.
Superintendent of State hospitals: £1,025 to £1,150 (less £100 for quarters).
Superintendent of large mental hospital: £1,050 to £1,150 (less £100 for quarters).
Superintendent of small mental hospital: £950 to £1,000 (less £85 for quarters).
Deputy Medical Superintendent of large mental hospital: £900 (less £60 for quarters).
Senior Medical Officer, mental or general hospital: £750 to £875 (less £60 for quarters).
Medical Officer, mental or general hospital: £600 to £700 (less £120 for board and lodging).
Junior Medical Officer, Education Department, male: £600 to £650; female: £500 to £540.
Medical Officer, Grade I, Education Department, male: £750 to £850; female: £620 to £700.
Oculist: £800 to £950.

The heads of the three services and the junior resident medical officers at the Coast Hospital (temporary appointees for one year) are not covered by the agreement.

Like all citizens of New South Wales, public medical officers are subject to State taxes, including an Unemployed Relief Tax of threepence in the pound, and like all New South Wales civil servants are at present subjected by a special Act of Parliament to a salary deduction of 8½% because of the state of the public finances.

Yours, etc.,

H. HASTINGS WILLIS,
Honorary Secretary,
Public Medical Officers' Association of New South Wales.

Sydney.
October 27, 1930.

THE LIFE OF SIR ASTLEY COOPER.

SIR: Could you help me in your columns to ascertain if any members have the "Life of Sir Astley Cooper, Surgeon," in two volumes, edited by his nephew, Sir Astley Paston Cooper, in the early 'forties. If so, I should like to communicate with them.

Yours, etc.,

BASIL FOULDS.

Medical Branch, Railways,
Central Station, Sydney.
November 13, 1930.

SIMPLE ACHLORHYDRIC ANÆMIA.

SIR: In the issue of this journal for October 18 you published a review of a most interesting article bearing the above title, which came from the pen of Dr. L. J. Witts, of Guy's Hospital. The syndrome described is isolated from the large group of secondary anæmias of middle-aged women by the fact that it occurs only with complete achlorhydria, frequently with glossitis and very rarely with splenomegaly. The glood picture resembles chlorosis and the Van den Bergh reaction is negative. It may follow pregnancy, is rarely progressive or fatal, and is unaffected by liver therapy. Witts, however, is convinced of a close relationship to Addisonian anæmia, either ætiologically or consecutively, and in particular to those anomalous cases of the latter disease which show a low colour index or an unsatisfactory response to liver therapy, and quotes examples of a familial incidence and association with the Plummer-Vinson syndrome. Some factor other than inadequacy of the usual liver hormone is obviously at fault in these cases, possibly a further hormone which is concerned in hæmoglobin manufacture rather than simple erythrocyte formation, also dependent on certain products of gastric digestion and not destroyed by the toxins of duodenal streptococci. Contrary to our reviewer's remarks, there is no evidence of either a "metabolic disturbance" or "visceroptosis" being ultimately concerned, and the pathology of the so-called primary anæmias was never so near a solution as at the present time.

The appearance of an anæmia of either a primary or a secondary type during or after pregnancy is perhaps related to the great demands made on the liver and its apparently more vulnerable condition at such a time.

Coincidently, the following two cases came under my care during the past six weeks:

(*a*) A.S., *ætatis* forty-four, seen in private, married, no children, thyreoid adenoma removed sixteen years ago. No previous illnesses. Sought advice for periodic attacks of diarrhœa and abdominal pain, alternating with constipation, beginning seven years ago. Was told five years ago that she was anæmic. All teeth removed sixteen years ago. Periods normal. Examination showed extreme anæmia, glossitis, a just palpable soft spleen, no nervous impairment, low blood pressure. Blood count: Reds, 4,180,000; hæmoglobin value, 42%; colour index, 0·5. All red cells very pale, showing marked poikilocytosis and anisocytosis, with moderate polychromasia, but no nucleated forms. Leucocytes, 10,480, with no changes in the differential count. Van den Bergh reaction negative. A careful search failed to reveal any septic focus, parasitic infestation or source of hæmorrhage. A month's treatment with iron, arsenic, dilute hydrochloric acid, aperients and "Lividex" liver extract has failed to improve the reticulocyte count, though the patient asserts that she is feeling much better.

(*b*) H.S., *ætatis* thirty-five, an out-patient at the Royal Prince Alfred Hospital, married, no children, has had shortness of breath and pain on left side of chest for eighteen months, but was ailing previous to this. No serious ilnesses before. All teeth extracted five years ago. Headaches every morning, wakes with puffy eyes, feet also swollen at times, no loss of body weight. Appetite good. Bowels constipated. Periods profuse and regular. No urinary symptoms. Examination showed her to be very pale, but not yellow. Apart from a few pus cells in the urine and a well marked glossitis, all the bodily systems exclusive of the hæmatopoietic were normal. The spleen was not palpable. The fractional test meal showed complete achlorhydria. The icteric index was not determined. A blood count gave the red cells as 4,310,000 per cubic millimetre with marked pallor, anisocytosis and poikilocytosis. Hæmoglobin value was 45% and colour index 0·5. No nucleated reds were seen. Leucocytes numbered 5,680, with no differential abnormality. Treatment on the same lines as above with substitution of eight ounces whole liver daily for the extract has so far failed to alter the count, in spite of subjective improvement.

Both cases are strikingly similar and neither has visceroptosis nor any suggestion of icterus.

These patients may prove to be examples of the chlorosis-like group isolated by Witts, for which the term "simple achlorhydric anæmia" could be reserved with advantage. The possibility of a transition of the blood picture to the pernicious type must be left open (especially in H.S.), but in both instances the anæmia has been known to exist for many years already. Perhaps a more experienced reader has had less difficulty in giving a prognosis in these cases.

Yours, etc.,

KEMPSON MADDOX.

141, Macquarie Street,
Sydney.
October 28, 1930.

Proceedings of the Australian Medical Boards.

VICTORIA.

THE undermentioned have been registered under the provisions of Part I of the *Medical Act*, 1928, of Victoria, as duly qualified medical practitioners:

Mancy, Ernest Henry, M.B., B.S., 1929 (Univ. Melbourne), 180, Camberwell Road, Camberwell, E.6.
Short, Oswald Victor, L.R.C.P. *et* S., 1930 (Edinburgh), L.R.F.P.S., 1930 (Glasgow), "Morningside," 43, Cochrane Street, North Brighton, S.5.

Additional diploma registered:

Hughes-Jones, William Eric Archer, M.D., 1927, M.S., 1929 (Univ. Melbourne).

TASMANIA.

The undermentioned have been registered under the provisions of the *Medical Act*, 1918, of Tasmania, as duly qualified medical practitioners:

O'Connor, Norman Joseph, M.B., B.S., 1924 (Univ. Melbourne), Waratah.
Duck, William Joseph, M.B., B.S., 1929 (Univ. Melbourne), Launceston General Hospital.

Books Received.

AN INTRODUCTION TO HUMAN EXPERIMENTAL PHYSIOLOGY, by F. W. Lamb, M.D., with a foreword by A. V. Hill, Sc.D., F.R.S.; 1930. London: Longmans, Green and Company Limited. Demy 8vo., pp. 348, with diagrams. Price: 12s. 6d.

DOCTORS AND SPECIALISTS: A MEDICAL REVUE WITH A PROLOGUE AND A GOOD MANY SCENES, by Morris Fishbein, M.D.; 1930. Sydney: Angus and Robertson. Crown 8vo., pp. 118, with illustrations by Dan Layman. Price: 5s. net.

Diary for the Month.

Nov. 25.—New South Wales Branch, B.M.A.: Medical Politics Committee.
Nov. 26.—Victorian Branch, B.M.A.: Council.
Nov. 27.—New South Wales Branch, B.M.A.: Branch.
Nov. 27.—South Australian Branch, B.M.A.: Branch.
Nov. 28.—Queensland Branch, B.M.A.: Council.
Dec. 2.—New South Wales Branch, B.M.A.: Organization and Science Committee.
Dec. 2.—New South Wales Branch, B.M.A.: Post-Graduate Work Committee.
Dec. 2.—New South Wales Branch, B.M.A.: Hospitals Committee.
Dec. 3.—Victorian Branch, B.M.A.: Annual General Meeting.
Dec. 4.—South Australian Branch, B.M.A.: Council.
Dec. 9.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
Dec. 11.—New South Wales Branch, B.M.A.: Branch.
Dec. 11.—Victorian Branch, B.M.A.: Council.
Dec. 12.—Queensland Branch, B.M.A.: Branch (Annual).
Dec. 16.—New South Wales Branch, B.M.A.: Medical Politics Committee.
Dec. 19.—Queensland Branch, B.M.A.: Council.

Medical Appointments.

Dr. R. Belli (B.M.A.) has been appointed Government Medical Officer at Tuncurry, New South Wales.

* * *

Dr. F. W. Cotton (B.M.A.) has been appointed Quarantine Officer at Esperance, Western Australia, under the provisions of the *Quarantine Act* 1908-1924.

* * *

Dr. I. G. Halley (B.M.A.) has been reappointed as an Official Visitor to the Mental Hospital at Parkside, South Australia, under the provisions of the *Mental Defectives Act*, 1913.

* * *

Dr. J. S. Smyth (B.M.A.) has been appointed Government Medical Officer at Warwick, Queensland.

* * *

Dr. J. K. Joyce (B.M.A.) has been appointed Government Medical Officer at Barcaldine, Queensland.

Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xvi.

CHILDREN'S HOSPITAL, INCORPORATED, PERTH, WESTERN AUSTRALIA: Junior Resident Medical Officers.

HOBART PUBLIC HOSPITAL, HOBART, TASMANIA: Junior Resident Medical Officer.

INFECTIOUS DISEASES HOSPITAL, FAIRFIELD, VICTORIA: Consultant Surgeon.

MATER MISERICORDIÆ PUBLIC HOSPITAL, BRISBANE, QUEENSLAND: Resident Medical Officer.

RENWICK HOSPITAL FOR INFANTS, SUMMER HILL, SYDNEY, NEW SOUTH WALES: Two Acting Relieving Honorary Physicians.

SYDNEY HOSPITAL, SYDNEY, NEW SOUTH WALES: Honorary Relieving Assistant Surgeon.

THE BRISBANE AND SOUTH COAST HOSPITALS BOARD, QUEENSLAND: Honorary Out-Patient Surgeon.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company, Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Members desiring to accept appointment in ANY COUNTRY HOSPITAL, are advised to submit a copy of their agreement to the Council before signing, in their own interests. Brisbane Associated Friendly Societies' Medical Institute. Mount Isa Hospital. Mount Isa Mines.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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